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THE DISEASES OF CHILDREN

A WORK FOR THE PRACTISING PHYSICIAN

EDITED BY

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WITH AN INTRODUCTION BY

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IN FOUR VOLUMES

*Illustrated in black and white and in colors by 61 full-page plates
and 430 text cuts.*

VOL. I.



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Electrotyped and Printed by J. B. Lippincott Company
The Washington Square Press, Philadelphia, U. S. A.

INTRODUCTION

BY

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A DISTINCTIVELY pediatric literature in the United States is barely a quarter century old. Previous to that time, what was written regarding children and their diseases was for the most part included with obstetrics or with general medicine, and the teaching of pediatrics, what little of it there was, was with one or two exceptions given by men occupying one or the other of these chairs in our medical schools.

One of the most striking things in connection with the rapid development of this special branch of medicine has been the growth of pediatric literature. The past twenty years have seen the establishment of two special journals, one four-volume cyclopædia, eight general text books, and at least a score of monographs or books upon special subjects.

The same period has witnessed the organization of a national society and many State, County, and city societies for the study of pediatrics.

It is interesting to see what has been accomplished in this period, —whether any results sufficiently important to be evident in mortality statistics have been produced. The study of the mortality reports from three fairly typical American cities is illuminating in this connection. I have selected New York, Rochester, and Yonkers. Reducing the mortality records of all these to a uniform scale, for the sake of comparison, we find that in New York City (Boroughs of Manhattan and Bronx), the mortality of children under five years, per 100,000 of population, has fallen in eighteen years from 1160 to 620. During the same period in Rochester, an inland city of 180,000, it has fallen from 584 to 340. In Yonkers, a suburban city of 60,000 inhabitants, it has fallen from 880 to 660. A calculation based upon the present population of children under five years, shows an annual saving of the lives of 12,000 children of this age in New York City alone.

There is, possibly, no branch of medical science which has developed more rapidly during the past two decades than Pediatrics; and it is to be doubted if there is any other which can point to such a record of achievements.

To what are these striking results due? To many influences, doubtless. But chiefly to newly acquired knowledge in the hygiene and medical treatment of children, in both hospital and private practice, and the dissemination of such knowledge through the agency of pediatric literature and the teaching of the medical schools.

What I wish now to emphasize is the important part played by pediatric literature in bringing about this result. As text books and medical journals have more readers than the medical schools have students, so is their influence much wider in spreading new and correct views in all departments of medicine. Every publication brings a new contribution, in so far as it chronicles a new experience; and the more widely such an experience has differed from our own, the greater may be its assistance to us. Although medical problems are much the same the world over, the conditions surrounding them differ greatly in different countries. The point of view also varies, and the emphasis is not everywhere placed alike. It is fortunate that this is so.

It is for this reason that we welcome the appearance of an English translation of a work of such scope as that of Pfaundler and Schlossmann, believing that it will be helpful to those who are treating sick children in private practice, and suggestive and stimulating to those following lines of research or engaged in practical work in hospitals and in teaching; and that its utility will be in no wise diminished but rather increased by the fact that its teachings differ in many respects from our traditional beliefs and practices.

PREFACE

THE present manual is designed for the use of the practising physician. It is intended to be his guide in the extensive and intricate province of infantile therapeutics, and to impart the knowledge necessary to him in the exercise of his profession with children both in health and in sickness.

Planned on a broader scale and more in detail than the ordinary text books, our work is meant to be a trustworthy book of reference, as well as interesting and instructive when read continuously.

Unhappily, it seemed impossible for a single member of the younger generation of specialists to describe fully and comprehensively, from his own resources, the whole range of infantile therapeutics. Such a task might be accomplished by the men who at this moment stand at the zenith of their productive powers, our teachers and the teachers of many of our fellow-workers, with whom and through whom our specialty has grown up; but our later generation falls far short of such omniscience. Our motto, then, must be, "Divide et Impera!"

We thought it a useful rule to intrust each chapter to an author who had devoted himself with special success to the subject of which it treats. Everywhere we met with a friendly response to our requests for assistance and coöperation, and with approval and support for our project. Many eminent colleagues, representatives of almost all the seats of investigation of children's diseases within the domain of the German language, entered willingly into the service of our cause. Our warmest thanks are preëminently due to all our associates in the work.

It was clear to us from the first that a division of the subject among a considerable number of authors would inevitably be attended with certain disadvantages. Occasional slight repetitions could not be avoided, and here and there somewhat different views of one and the same question have been taken. But there are very striking and decided advantages in such a division of the work. Each writer is able to present to the reader in a concise form the particular province which he has mastered by personal investigation. Furthermore, continual changes in the method of presentation and in style tend to render the perusal more interesting.

The editors had to establish first the outlines of the building and then to join the well-hewn stones into an harmonious whole. We have judged it best, in a book intended primarily for the needs of the practi-

tioner, to limit the pathological anatomy to the most important facts, and to put the physiological part in the foreground, together with the modifications resulting from the lesion.

The sole responsibility for the form and contents of the different articles rests, of course, upon their respective writers.

The methods of instruction in our clinics have been supplemented in many technical ways, in order to assist the memory and understanding of the student. This is the result of the continual opening of new fields of knowledge. One such aid is that of graphic representation. We have therefore taken pains to make the illustrations as complete as possible, in the firm belief that a good picture conveys more and makes a deeper impression than does many a long exposition.

The difficult task of procuring proper subjects for reproduction has been lightened by the kindly aid of many colleagues, especially by the heads of clinics and hospitals for children.

Part of the colored plates have been reproduced from moulages which were prepared, for the most part, by Mr. Fritz Kolbow, the manager of the pathoplastic institute at Berlin. He aided us also with the reproductions which the Dresden firm of Roemmler and Jonas has executed so excellently. Other plates have been produced by a process which is here employed in practice for the first time; namely, by colored reproductions made directly from fresh or properly preserved preparations.

Behind us lies a period of earnest application to the now completed work. We have watched its growth and development with joyful expectation, and we send it forth in happy confidence. May the profit to the public be the reward of our zealous coöperators and of ourselves.

M. PFAUNDLER
A. SCHLOSSMANN

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INTRODUCTION

BY

PROFESSOR A. SCHLOSSMANN, OF DÜSSELDORF

ὁ περὶ παιδοτροφία λόγος ἐστὶ
μέγα πλατὺς καὶ πολυμερής.

—SORANUS OF EPHEBUS, "*De arte obstetrica morbisque mulierum quæ supersunt.*"

A BIOLOGIC LAW teaches us that the excessive growth of any organism leads to its division. In an analogous manner, the growth of knowledge in the wide field of natural sciences has caused it to split into various branches.

That branch which deals with the treatment of man in health and sickness, Medicine, is our basic science. The more the knowledge of physiological and pathological facts increases, the wider is the experience which must be added to the physician's knowledge and skill. As a result, the general science of medicine separates into a number of special branches. These, in constant touch with one another, strive upward as individual shoots, but their roots cling to the common soil.

Infantile therapeutics has also become a special science. No one can become a general practitioner, in the true sense of the word, without giving it very earnest attention. So, on the other hand, no physician can make children his specialty, whose special knowledge does not rest on the broad basis of general medical science. He must be thoroughly familiar with physiology, pathology, hygiene, and internal medicine, for without such ground-work, he is not worthy of the name of specialist. The right of infantile therapeutics to be considered a special branch of medicine is no longer contested. The attempt to do so would be stamped immediately as showing a fundamental ignorance of pediatrics and its significance.

The causes which led to the independence of our science are to be sought in the science itself. Internal medicine, in general, deals with the changes in condition which take place in the adult, the mature man, under the influences which originate disease. The phenomena of disease, though the disturbing causes are alike, follow a different course in the nascent man, the child, whose development has not yet reached its term. The provocative which produces the same disease in young and old is indeed the same, but its effects vary in intensity and its course is different.

Especially is this true in the earliest stage of life, the nursing period.

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Here we find the acclimatization to the exterior environment, the extra-uterine life; and we have to deal beside, not with the omnivorous adult, but with an individual feeding exclusively on milk.

But it would be a great mistake to consider the nursing period as the only one coming within the special province of infantile therapeutics. The age before the school duties begin has manifold dangers and forms of disease peculiar to itself, of which I will mention only rachitis and scrofula. Then comes the school age, with the changes in condition caused by the school itself, with the appearance of the contagious and infectious diseases which are emphatically designated as diseases of children. And, finally, we have the age of sexual development, which may likewise be attended with very special symptoms. All these stages in the process of man's development offer abundant opportunity to the physician to convince himself of the peculiar reactions, very different from those observed in adults, which the organism of the child exhibits in response to external influences. Almost every page of this book gives proof and evidence that great numbers of scholars have applied themselves with tireless energy to do justice to the special needs of childhood.

If we let our mind roam backward over the last twenty-five years, we shall see an astonishing development in infantile therapeutics. Henig wrote in Gerhard's manual an article on the history of infantile therapeutics, tracing it back to the earliest times. Shortly after the appearance of Gerhard's great work a number of members of the pediatric section, at the fifty-fifth meeting of the German naturalists and physicians in Eisenach, met together to establish a Society for Infantile Therapeutics on the broadest of foundations. This plan was carried into effect in the following year (1883), at Freiburg im Breisgau, and over one hundred German speaking physicians who were interested in the subject joined the new association.

Since then the Society for Infantile Therapeutics has exercised a stimulating and decisive influence on the development of our specialty. The number of physicians taking part in the annual discussions has increased from year to year, and no one of them can have gone away without a touch of inspiration. First under the presidency of the tireless Steffen, and now under that of Heubner, the customary discussions have faithfully reflected the important progress which infantile therapeutics has achieved.

Many auxiliary sciences have been forced to enter the service of their pediatric sister. Clinical observation, which by the aid of physical methods of investigation had attained the flower of classic perfection, no more sufficed alone. The etiology of a series of important diseases was explained at this time by Robert Koch. The specific generators of many infectious diseases to which children are especially liable are still unknown at the present day, yet the influence of the new etiological

knowledge on our entire practice as physicians is very striking. The development of the doctrine of immunity, and its well-planned transformation from a theory to a practical system, the struggle against diphtheria by means of the specific curative serum, which we owe to Behring, must rank among the most beneficent achievements of mankind. One of the most fatal of all diseases, one which loved to seek its victims among children, has been robbed of more than half its terrors.

The results obtained here necessarily awaken hope of like advancement in neighboring fields. In no case so far has the victory over other infectious diseases been won so decisively, yet there is much to show the possibility of future success.

Beside the use of the serum, still another way has been opened to strike at the root of disease. I refer to the specific benefits obtained from the extracts of various organs and from nourishing the organs themselves. To appreciate the importance of these methods it is enough to bear in mind the favorable influence exerted on myxœdema through organotherapy. In those cases where we are unable to reach the disease and cure it through its causes, pharmacology affords us means or alleviating the symptoms. No doubt there lies some danger of injury through the multiplicity of remedies, through a too copious and a vacillating employment of all the means supplied by modern synthesis. But there a sound judgment must point out the proper path. Now we may alleviate pain where formerly we were helpless, and may bring relief to the suffering child in many ways, where once the task was beyond our power.

The persistent endeavor to utilize the results of modern physiological ideas in infantile therapeutics has played an important part in shaping its development. It was the senior Camerer, at first lightly esteemed but now generally recognized, who pointed out this path to us and who gave the theory of metabolism and the laws of energy an appreciable part in our physiological scheme.

The possibility also of observing the diseases under more favorable conditions has been increasingly impressed upon us by the improvements in hospitals for children. Within the period which we now briefly review, the last twenty-five years, are embraced the endeavors to establish model institutions (Leipsic, Berlin; Baginsky's Kaiser und Kaiserin-Friedrich Kinder Krankenhaus, etc.) designed to limit to the minimum the transmission of infection from child to child. The teachings of antiseptis were, *mutatis mutandis*, confined for the main part to the treatment of patients suffering from acute infectious diseases, in order to prevent the spread of the disease within the hospital itself.

A peculiar interest, especially within the last ten years, has been accorded to nursing infants. Their nourishment, their care, their shelter in health and sickness, has been the subject of continuous and varied investigations and fills a large place in pediatric literature.

Many universities still lack adequate facilities to insure profitable instruction in pediatrics, and in many cases have no instructors fully conversant with the progress of our science.

Rightly did Escherich predict an imminent change in this direction when he called attention to the appreciation which infantile therapeutics finds in the most modern seats of investigation and learning and at the schools of practical medicine. With joyful assurance, therefore, we look forward to the future of our science. Its practical importance, as well as the zeal of its disciples, insures its progressive and successful development.

THE DISEASES OF CHILDREN

GENERAL PATHOGENESIS AND PATHOLOGY OF CHILDHOOD

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IN the study of Pediatrics we see that along certain lines the child is distinctly different from the adult. The child reacts to the same disease in a different manner from the adult. Certain diseases predominate during childhood rather than later, and the same disease in the young person is often seen to run a markedly different course from that in later years. To obtain a successful insight into the pathogenesis and pathology of this period one must become definitely acquainted with the peculiarities of the child.

Before we speak of these idiosyncrasies, we must first have clearly in mind certain general considerations relative to disease and its etiology.

Definition of Disease.—Disease is understood as the reaction of the body to a specific exciting cause. In man this really depends not merely on a single reaction, but always on the sum of many reactions (*i.e.*, the reactions of the different organs which are affected by the disease). The individual reactions of the different organs, or cells of the organs, produce a symptom-complex, which represents to the mind of the physician a clinical picture of some definite disease. As each disease depends on the reaction of the individual cells, careful investigation of the vital processes of each cell would give us more fundamental and definite ideas on the course, cause, and specific organism of the disease.

The health of each cell or group of cells is intimately dependent on certain conditions most favorable for life. It thrives best at certain temperatures, under certain atmospheric pressures, and in a certain chemical relationship to its surroundings. Any change in these conditions acts as a stimulus for the cell. As long as the stimuli keep within certain bounds, no perceptible variations are seen in the normal life of the cell, and the cell remains healthy. When, however, these changes

exceed certain limits the stimuli become strong enough to produce a change in the cell. These changes are recognized by some disturbance of the normal physiology and are known as "disease." So long as the changes in the conditions of life do not produce disease or sickness they are considered *physiological stimuli*. When they pass this physiological point and cause disease the stimuli are said to be *aphysiological* or *pathogenic*.

The greater the variations in the environments of a cell or its forebears, the stronger can be the stimuli without making such a cell diseased. On the other hand, a cell whose progenitors have always lived in uniform environments will feel a very slight change of the surroundings as an aphysiological stimulus. Thus the origin of a disease is always an aphysiological stimulus, one to which the cell is unaccustomed and against which it is not strong enough to fight.

It is comparatively easy to understand what are the best environments for a unicellular organism; what is a physiological and an aphysiological stimulus, or what represents disease for the cell; but it is a much more complicated matter to find out the same things for the human body. This depends on the fact that each cell of the body is widely different from the others, and has a markedly different sensibility to the same disease. Each group of cells is further seen to be very dependent on the others, so that a slight change in the one will call forth at times a severe disease in the other. In this connection, attention should be called to the importance of the internal secretion of certain organs (the lymphatic nodes, adrenals, etc.).

The Importance of the Epithelium.—The cells which play the most important part in the appearance of a disease are those found on the external and internal surfaces of the body.

The Epithelium.—Coming into contact with the outside world these cells are naturally more accustomed to changes in their environment, and by their action maintain the normal uniform conditions necessary for the existence of the deeper cells. The most important epithelial organs are the skin, the digestive system, and the respiratory system.

The cells of the skin are accustomed to fluctuations of temperature to which the deeper cells are not. Thus we see that the skin, which is able to obstruct the entrance of foreign bodies, can also keep the temperature of the internal organs at a uniform degree. It accomplishes this by certain mechanisms, but only so long as the variations of external temperature are not too great.

The *epithelial cells of the digestive tract* prevent the entrance of bacteria and foreign albumins, by means of their digestive power. Thus, in spite of their permeability, which they must have in order to absorb food, they form a protective apparatus of extraordinary value.

The *epithelial cells of the lungs* allow the interchange of oxygen and carbonic acid gas, but cannot of themselves prevent the entrance of foreign bodies. Such substances are caught by the to and fro motion of the ciliated epithelium of the upper air-passages. Warming the air as it passes through the nose, larynx, and trachea is another duty of the epithelium, which thus guards the organism from a too great reduction of temperature in the act of breathing.

Naturally, this function of the epithelium depends on certain external conditions. So long as the variations of these conditions are not too marked,—in other words, so long as the external temperature and the atmospheric pressure fluctuate between certain limits, so long as the dust and bacterial content in the air is not too great, and so long as the nutrition is good,—the body will remain healthy. When the external conditions deviate to any extent and become aphysiological, the epithelium becomes diseased and may even die. The epithelium will then be an insufficient protection, and substances can enter and do harm.

The Cells of the Deeper Tissues.—Cells of the deeper tissues react to slight variations of temperature and changes in osmotic pressure, because such deviations are unnatural to them. The skin should keep the temperature normal, while the intestines and the kidneys should be able to keep the osmotic pressure in the tissues at customary height. These cells possess as well a definite chemical constitution, which produces a peculiar chemical reaction in the blood, lymph and other fluids of the tissues. Thus, a slight change in the chemical constitution of the blood will produce a severe aphysiological irritation for these cells.

Each organism tries to keep in every way possible a fairly uniform temperature, definite osmotic pressure and chemical constitution for its cells and tissues. When this cannot be maintained, conditions arise which injure the tissue-cells. While the epithelium is acting normally, the body will be healthy. When its functions are disturbed by outside changes, it becomes sick. This is explained primarily by the direct influence of the affection on the skin, and secondarily by the entrance of harmful substances which should normally be arrested by it, etc. For example, when the outside temperature drops below the ordinary endurance of the skin, the entire body suffers from cold, and death is produced by freezing. The latter conditions are seen when the low temperature injures the mucous membranes, say of the respiratory tract, and makes it sensitive to bacteria, which thus secondarily produce disease (coryza).

From this it is seen how injuries of the epithelium often form the predisposing cause for a disease. They are changes in temperature, trauma, dust and bacteria in the air, and finally a previous faulty nutrition. This latter may arise from too much, too little, or perhaps improper food.

The results of injury to the epithelium are usually seen in the penetration of foreign substances into the deeper tissues of the body. Here they call forth a reaction, being aphysiological stimuli. These invading substances may be destroyed by this reaction or again appear as a disease, more or less severe, when the destruction does not proceed smoothly.

Bacterial Causes of Disease.—Experience teaches that the substances which pass through the epithelium and produce disease in the inner organs are almost exclusively protozoa and bacteria. Now, we know that certain bacteria do not produce disease in man, even when they enter the deeper tissues. Again, there are bacteria which, pathogenic to man, do not give a disease when injected into other species of animals. That certain bacteria always produce disease in man but do not show any reaction in other animals is explained by the following: These bacteria meet in the cells of the human species substances which do not exist in the other species, and the consequent reaction produces disease. This peculiarity of reaction is called the *Idiosyncrasy of Species*.

Since most bacteria produce no disease when they enter the body in small numbers, it is supposed that they die, and that this death is dependent on the reaction produced by the presence of the bacteria in the tissues. In order to produce a disease it is essential for the bacteria entering the body to meet cells which will combat them. These cells in the human species are the leucocytes, either the polyleucocytes or the monoleucocytes. This idea of resistance agrees with the phagocytic power of the leucocyte, discovered by Metschnikoff and Hans Buchner. The leucocytes are the only cells of the body which retain the peculiarities of the amœba, as to migration and digestion. This fact explains certain things we have not been able to fathom. "Why is it that infectious diseases of the respiratory tract are often accompanied by a high leucocyte count, as in purulent catarrh of the upper air-passages, bronchitis, and pneumonia, while almost none are seen in diseases of the digestive tract?" The answer to this question is that the cells of the digestive tract are able to protect themselves unaided, while those of the respiratory tract need the leucocyte to help them resist.

The human cell must, by its very nature, react to bacteria by producing disease when they enter the system, in the same way that the muscle-fibre is forced to contract in reacting to an electric stimulus. The amount of the specific cause of a disease (contagium), be it syphilis or measles, does not matter. The patient will in any event fall sick, even though the quantity be small.

Not only must mankind react to a certain distinct etiological factor by the appearance of a disease, but this must be a *certain definite disease*. Men, being similar to one another, must of necessity react to the same stimulus with approximately the same objective and subjective symptoms, thus making it possible for us to diagnosticate a disease. Dif-

ferent individuals of the same species present a slightly different reaction as a result of the same disease, because of the slight difference of their cells, and in the same way small differences in susceptibility are seen in the same family. We can carry this still further by saying that we may have an *individual idiosyncrasy* to disease as well as a racial and family one.

The reason why the different organs of the same species react differently to the same disease is that in spite of their typical similarity to each other they are differently constructed, according to their different functions. So we speak of an *organ-disposition*. To illustrate, the gonococcus produces ordinarily an inflammation of the conjunctiva; joints, and lower urinary passages; the diphtheritic organism affects the tonsils and upper air-passages; while the specific cause of mumps produces an inflammation merely in the parotid gland and its immediate vicinity. These phenomena depend, we imagine, on a rather definite selective power, the actual existence of which is still undemonstrable. It is supposed to be a power of attraction existing between the bacteria and their toxins for the cells especially affected by these organisms.

While the cells of the different species, as also those of the different organs, have a slight variation one from another, we see that they also differ according to the age of the individual. This *idiosyncrasy of age*, intimately connected with the idiosyncrasies of race and organ, helps to explain why the cells of a child react to a stimulus in quite a different manner from those of the adult.

The *normal disposition* we consider to be that peculiarity of the healthy individual to respond to a definite etiological cause by the appearance of a particular disease. If we believe this, then it must naturally be concluded that there may exist constitutional tendencies more or less different from the normal, which, reacting to a stimulus normally physiological, produce a disease. This increased susceptibility to a disease is influenced by external conditions, not only those which aid in its appearance, but also those which have an unfavorable influence on the course of the disease. Such hypersensitiveness to special diseases may be inherited, or may be acquired from unfavorable hygienic conditions, dependent on poverty, mistakes in feeding, and other diseases already existing. A single harmful influence may of itself be unable to produce this condition, whereas in combination with others, disease would readily appear.

There are many things which in a general way help to answer the question as to why certain disorders often appear in childhood, or at least seem more prominent at this time. Acute infectious diseases, for instance, seem to predominate in childhood because the adult is immune; having already experienced them. Again the imperfect development of certain organs in the child, as compared with the same organs in later

life, offers a reason for the frequency and different course of certain other diseases seen at this time. Not only is the period of childhood a factor in disease, but the period of adolescence also, and certain outside conditions as well.

Acute Infectious Diseases.—We will speak first of the acute infectious diseases peculiar to the child, by the laity called the “diseases of childhood.” Most infectious diseases are more frequent in childhood because, in contrast to the adult, children possess an especial susceptibility, due to their age. The fact that the first attack of these diseases usually makes a person immune, and the fact that children are especially prone to them, explain still further why they appear much oftener in childhood than in later life. This frequency depends not only on a disposition of age but on a racial disposition; that is, the fact that all mankind is susceptible to these diseases. To this group of diseases belong measles, rōtheln, scarlet fever, and whooping-cough.

The tendency to catch scarlet fever and whooping-cough, while it is less in the adult than in the child, still remains throughout life. In contrast to this we see that the peculiarity to take chicken-pox and mumps, so marked among children, is not seen in adult life, although the individual may not have experienced them during earlier years.

All the diseases above mentioned, with a rare exception, give prolonged immunity; that following diphtheria, however, is quite often of short duration. It is not known whether this depends on a disposition peculiar to the individual or on an abnormally rapid disappearance of the antitoxin.

The origin of acute infectious diseases is the presence of the contagium or exciting cause, which remains on the mucous membranes of the mouth, pharynx, and throat of the child. If present in sufficient amount this contagium, after its incubation period, produces the disease characteristic of it. Every infectious disease has a more or less definite type of infection, which depends, first, on the peculiarity of the exciting cause, and second, on the disposition of the patient. To illustrate: in measles the contagium is supposed to live outside the body only a very short time, so that the rapid spreading of an epidemic can only be explained by the extraordinary high susceptibility of man. Its transmission almost exclusively arises from direct contact. Quite the opposite is seen in scarlet fever. The exciting cause in this instance remains active for a long time outside the body and is still able to produce infection. So that, in contradistinction to measles, the susceptibility for scarlet fever is not so great.

A fairly good comparison can be drawn between measles and syphilis on the one hand, and scarlet fever and tuberculosis on the other. Infection to the former is governed by the very slight power of resistance to the contagium while outside the body and the high susceptibility of

man; by the latter, by the high resistance to the exciting cause exerted by a disposition less susceptible.

We have just seen how nearly every acute disease leaves behind it an especial immunity. It is most surprising, then, believing as we do that this immunity depends on the presence of an especial material in the blood (a humoral immunity, or cellular immunity of the organism not clearly understood), that the child shows a tendency to catch the disease against which its parents have already acquired immunity. The child inherits peculiarities of race and species as well as those of the individual parent. Why then are the powers of immunity not also inherited? We know that its parents are immune, but notwithstanding this, nearly every child readily catches scarlet fever and measles. The answer to this question is readily made by deduction and is of importance to the far weightier questions of transmitted and inherited disease, as well as to that of inherited predisposition.

At the outset, we must accept the fact that the generating cells of the father and mother contain not only the characteristics of the species, race, and family, but the individual peculiarities of the parents, as well. If all the characteristics of the father and mother are present in the foetus,—a product of the sexual cells in the process of generation,—and if all these peculiarities, dependent on the definite arrangement of cell protoplasms, have been transmitted to each individual cell of the child by the assimilation of nourishment, then it is conceivable that the child must resemble the parents, even in their individual characteristics. According to this, every characteristic of the parent should be transmitted, and the child should thus possess an immunity through that of its parent, *i.e.*, against scarlet fever. This power, however, is assumed only by the leucocyte, for, as mentioned above, this is the only cell which reacts to a disease. This explains how the human body especially provides protection against the transmission of acquired diseases. Most of the bacteria, and especially their toxins, react with the leucocytes (as seen in the Introduction). The spermatozoon and the ovum are not affected, as they never come into contact with the other cells, and so their protoplasmic structure is unable to assume a special immunity, and it is therefore impossible for the germinating cells to give the organism (*i.e.*, the child) immunity.

The law that only those characteristics can be transmitted from the parent to the child, which are somehow contained in the germinating cells, is of much importance to our study of pathogenesis and pathology. The idea so clearly explained by Martius strengthens this assertion. He says, in a few words, that there is absolutely no transference of a disease from a parent to a child. Disposition or tendency for a disease is the only thing which can be transmitted from the parents. This tendency or susceptibility has its foundation in the definite composition

of the cellular protoplasm of the human being and so must be present in the cells of germination as well. While susceptibility for a disease can be inherited, the disease itself can never be, for the disease is a fact, not a characteristic or a condition. Disease is easily transmitted from the mother to the child, but it can never be inherited as such.

The same thing may be said of immunity: it can never be inherited, but only transferred from the mother to the child. There are many instances in experimental pathology in support of this fact. Immunity depends therefore on a transfer of some antibody from the blood of the mother into that of the child,—a passive immunity, produced by the protective qualities of the mother-blood. It is always a hæmatogenous and never a cellular immunity.

Since acquired immunity against certain infectious diseases depends not only on an increased resistance of the cells, but also on the presence of certain protective materials in the blood, we can easily imagine that the newborn infant in the first weeks of life possesses an immunity against diseases already experienced by the mother. Perhaps this fact explains why babies in the first weeks of life do not catch measles, scarlet fever, whooping-cough, or chicken-pox. Just so far as immunity is "in-born" and not inherited, are there inherited diseases. We can speak of "innate," or, still better, "intra-uterine" disease, but it is nonsense to speak of "inherited" syphilis. Let us here speak of the so-called "inherited syphilis" there being no syphilitic disease of the mother; by which we mean that the child is syphilitic, while the mother, showing no signs of the disease, is assumed to be free from it, and the child is supposed to "inherit" the taint. This old and improbable idea, depending on the just as improbable hypothesis of germinal infection, has been very energetically fought in the last few years by Matzenauer. This writer has concluded, from the most logical reasoning and from clinical observations, that there is absolutely no syphilis in the child without there being syphilis in the mother.

Those diseases which we call in-born are merely the results of disease contracted *in utero*. Anomalies of the heart and certain agenesis and hyperplastic conditions are either the results of some abnormal topographical conditions in the uterus (*e. g.*, amputation by the cord), or of injuries during labor. Many malformations are correctly understood to be dependent on the transmission of a pathological origin (as harelip and supernumerary fingers or teeth).

The newly-born infant, normal throughout, differs from the adult, as already shown, by its undeveloped condition. It reacts quite differently to the same surrounding conditions, and in a relative way to the origin and cause of disease. According to Escherich, one can differentiate certain periods in the life of a child by the irregular progress in the growth of the above-mentioned undeveloped organs of the infant. In each of

these periods the child will show a different susceptibility to disease. It will be best, in speaking of the pathogenesis of each period, to retain the usual classification. This is the one which Escherich also used in his studies on the morbidity of different ages. We shall speak of an especial pathogenesis for the newborn, the "suckling," the period of the first dentition (milk teeth), the period of the older child, and that of puberty. We will try to discuss pathogenesis from these points of view, and, as we have done in the Introduction, give the distinguishing features:

Diseases of the Newborn.—The diseases of the newborn, during the first week or two of life, are really little understood. In this short time an exact picture of disease can scarcely be formed, so that it is usual to lay the cause of death on the undeveloped condition of the infant, unaccustomed to extra-uterine life. This is a perfectly fair conclusion, too, as the first two or three days is not a long enough interval for an infection which might have occurred synchronously with birth to produce death. According to the statistics used by Eröss, as an illustration of the infant mortality during the first four weeks of life, almost one-half of all children die within the first week, and more than one-half of these succumb by the first or second day. While we may have rightly concluded that the main cause of this high mortality is the undeveloped condition of the newborn child, we must not forget one of the most frequent diseases of this period. This has as its origin an injury of the navel, which one may call physiological. In the falling off of the cord bacteria may enter the navel and produce a severe infection.

Premature Babies.—Before taking up the pathogenesis of the nursing child ("Säugling"), we must first speak of the premature baby. The most important diseases that affect infants of this age depend on the child's not being accustomed to the demands of extra-uterine life. Such children, accustomed to the conditions of intra-uterine life, feel extra-uterine existence as an aphysiological stimulus. Their power of resistance is so lowered in this way, that the slightest disturbance may cause death.

The Nursling ("Säugling").—In the Introduction we saw in a general way the importance of the condition and function of the skin and of the mucous membrane of the respiratory and digestive tracts. The skin of children at this age is much more delicate than later, and so is less resistant to injuries. Trauma nevertheless plays no especial part, as the nursing child is usually carefully protected by its mother from any possible harm. Infants, however, show a general susceptibility for eczema and intertrigo, as a result of the decomposition of the urine and stools. Furunculosis, also, which so frequently appears among infants, is caused, at least in part, by the sensitiveness of the skin. Other circumstances also, the chief of which is a generally lowered power of resistance, promote this tendency to furunculosis. Such general circum-

stances as artificial nourishment, faulty feeding, dirt, and unhygienic surroundings tend to decrease the resistance. Other diseases are seen which depend on a markedly increased sensitiveness of the skin, called by Czerny the exudative diathesis. Such diseases appear widely spread over the body and show themselves in the superficial layers of the skin as a tendency to eczema, etc.

The chief task of the skin, as we have seen, lies not only in protecting the body from the mechanical entrance of foreign substances, but also in keeping the temperature of the body at a certain degree, in spite of the variations of the outside air. The skin is thus an organ which acts as a heat-regulator for the entire organism, and which in adults works with extraordinary exactness. In the nursing infant this regulation of the temperature by the skin is moderately difficult, especially because the body surface in the child is relatively greater than it is in the adult. As a result of the cooling or overheating of the surrounding air, a very sudden lowering or raising of the body temperature may occur. Consequently, babies get very easily chilled, if the surrounding temperature is too low, especially in the first weeks of life. Moreover, they exhibit a very rapidly rising temperature if the air is too warm. While this fact supports the theory that the vasomotor power of the skin, controlling the temperature of the nursing infant, is small, we also find that in febrile diseases the temperature mounts very high, because of the contraction of the vessels of the skin, which makes a great effect, owing to the relatively great body surface.

The picture of still another disease, sclerema, depends, in part at least, on the above causes,—the relatively greater body surface of the infant and the resulting difficulty of maintaining an exact regulation of the body heat. In consequence of a pronounced lowering of the temperature, a partial stiffness of the subcutaneous fat takes place, which is due to the fact (shown by Knöpfelmacher) that the fat in the tissues of an infant has a higher melting-point than have the fats of the adult, owing to the small amount of fatty acids present.

What has been said in regard to the skin of the nursing infant, has been accepted for some time in relation to the mucous membranes as well, which are weaker than are those of the adult, and more sensitive, so that they cannot act as they should. While this hypothesis has certainly been proved true of the skin, it is not so of the mucous membrane, and one can merely speak of an especial sensitiveness of the mucous membranes of the infant. We think that this sensitiveness of the respiratory tract tends to catarrh, pharyngitis, bronchitis, and bronchopneumonia. This is borne out by what was mentioned in the paragraphs on general pathogenesis.

Diseases of the Digestive Tract.—Of the different affections seen among babies, those of the digestive tract stand out most promi-

nently, for the reason that no other organs of the child are so undeveloped. The digestive tract of the infant is dependent on a particular kind of nourishment,—woman's milk; and even with this nourishment, which represents certainly the best possible for the digestive cells of the baby, derangements of the intestinal tract appear. They are of a much milder type, however, than those seen in other cases. Profuse bacterial life, to which the intestine of the infant is not accustomed, forms substances in the chyme which irritate the digestive cells and produce catarrh. As a result, an abnormal production of gas stretches the intestine and causes pain.

Artificial Feeding. — Disturbances of digestion occur much oftener in artificially nourished infants than in those naturally fed. People have tried to explain in different ways this frequent appearance of intestinal troubles and their results in the artificially fed baby. Some have said that they depend on the different composition of woman's and cow's milk, especially on the richness of the latter in the casein element. Even with a modification of cow's milk resembling perfectly that of woman, with the proper intervals of feeding and the physiological quantities of food, there always appear relatively more derangements than when the child is fed on human milk. One can only conclude from these facts that cow's milk is so different from that of the mother that one may never hope to make the one in any way equal to the other. The foreign ("artfremde") albumins of cow's milk irritate the mucous membrane of the intestine of the child. This unaccustomed and thus aphysiological stimulus, damaging the digestive apparatus through continued use, indirectly affects the whole organism. What other things take place our imagination can well conceive. Schlossmann is correct in saying that cow's milk is an unnatural food for the infant. This unnatural and aphysiological stimulus harms the baby and especially its intestine, which not yet being fully developed, easily gets deranged. Such derangement happens oftener in the artificially fed than in the breast-fed child. One does not go far astray in calling the direct and indirect results dependent on cow's milk the "cow's milk diseases." Although cow's milk can seldom if ever be said to be the direct cause of death, it is doubtlessly a great contributing factor to the high mortality seen during the first year of life. We can readily see how cow's milk is a predisposing cause, for it may lower the normal resistance not only of the digestive tract but even of the entire organism. Many examples support this idea. Furunculosis is an example of a disease, the frequent appearance of which is due to the lowered vitality consequent upon artificial feeding.

We should like to take this opportunity to say that we do not consider that the only harmful influence of artificial food lies in the difference of the albumins. The varying percentages of the elements,

especially of the fat (particularly maintained by Czerny), play a prominent part in the injury, without taking into account the different bacteria due to the dirt in the milk. So long as we know so little of the quantitative composition of these elements in milk, we can say merely that the injurious results of artificial feeding are produced by the physiological stimulus of the milk of other animals. It is utterly impossible to copy the physiological stimulus of the mother's milk by means of other foods.

From what has been said in the Introduction, on the importance of the epithelium in protecting the health of the entire organism, the question naturally arises whether the mucous membrane of the intestine, which is so often injured by the action of cow's milk and therefore weakened, does not frequently allow bacteria to enter the internal organs, where they may develop and by their action produce further harm. That it does seems probable from the investigations of Czerny and Moser.

Tuberculosis.—The weakness of the intestine offers during the nursing period of a baby a possible cause for the appearance of one of the greatest of human scourges, tuberculosis. As Behring has expressed it, tuberculosis in the majority of cases, although it appears in late life, is dependent on the fact that tubercle bacilli at some time in earlier life have passed through the delicate wall of the intestine. In the intestine they have either produced a tuberculous lesion at once, or, lying dormant, have awaited a chance to reappear in later years. The intestinal wall of the nursing infant is asserted by Behring, from the experiments of Disse, to be more pervious to tubercle bacilli than is that of the adult. Disse is said to have proved, in the newly-born individuals of different species, that the mucous layer which lies on the epithelium layer of the mucous membrane, and which is unbroken in the adult, in the newborn is sieve-like. These openings may make it possible for the tubercle bacilli to pass through, and so in another way may explain the early origin of tuberculosis. It is by no means certain that these conditions are as simple as they seem. In the first place, the findings of Disse have not been proved by others. In the second place, no one has ever proved that the mucous layer is the chief protecting element of the epithelium.

Those who support the theory that tuberculosis is transmitted through the air, point to the following fact as strong evidence. The greatest changes following an infection of certain bacteria, as well as the tubercle bacilli themselves, are found in the glands of that region in which the infection takes place. Authors who have been studying the frequency of tuberculous changes in the definite groups of lymph-nodes, find that the bronchial nodes in the majority of cases are the ones chiefly affected, showing tuberculous changes of pronounced caseation. This

fact would seem to show that the tubercle bacilli in all probability have passed through the mucous membrane of that part of the body (the respiratory tract) into the bronchial lymph-nodes. According to the findings of Weleminsky, it is apparently proved that the bronchial lymph-nodes occupy quite a definite position in the topographical system of the lymphatics. He maintains that they drain the lymph streams of the neck as well as those coming from the intestine. On the other hand, Bartel has shown that the bronchial lymph-nodes are differentiated from the others by their internal structure. The bacilli which pass the imperfect filtering system of the cervical and the intestinal nodes are stopped by the bronchial lymph-nodes. The caseation of the bronchial nodes is explained by means of the combined streams of bacilli which have passed through the mucous membrane of the mouth, throat, and intestine. Bartel has also proven that the normal mucous membrane of the intestine, even in the adult, may be pervious to the tubercle bacilli.

While these facts support the possibility of tuberculous infection by the mouth, throat, and the mucous membrane of the intestine, the theories defending the idea of an air-infection, as the probable important origin of tuberculosis, are also sound. Certainly the fact that the bronchial lymph-nodes on the right are so much oftener affected than those on the left, seems to us to offer good evidence for the importance of an air-infection. The right bronchus must allow a greater amount of dust and bacteria to enter the lung than does the left. In the first place, it has a greater cross-section than the left, and in the second it runs more nearly parallel with the trachea, the left leaving the trachea at a comparatively sharp angle. Still another argument, and one not well known, is the following: Tubercle bacilli when on the mucous membrane of the bronchus are a greater danger to the organism than when on the mucous membrane of the intestine. While on the one hand it is a normal task and function for the mucous membrane of the intestine to digest and destroy bacteria, on the other, the presence of only a few bacteria is a very powerful aphysiological stimulus to the bronchial mucous membrane, which is normally accustomed to air well purified by the ciliated epithelium of the upper air-passages. Thus it seems to us more plausible that an infection with tuberculosis takes place more frequently through the air, than by the intestine. This seems true to us even though children take in more bacilli by the digestive than by the respiratory tract.

Another factor must be considered in relation to the appearance of tuberculosis; namely, what is called by Behring the "defect" healing of the tubercle. When the tuberculous lesion is entirely healed, instead of an increased resistance against tuberculosis being left, we see a greater sensitiveness remaining. A tuberculous lesion may disappear entirely,

but it accordingly leaves in the individual a susceptibility for the tubercle bacillus, which on a second infection by air, produces a slow-healing tubercular process.

In this connection, Baumgarten's theory of placental infection is important, especially so, as Schmorl and Geipel have for the first time shown that the possibility of placental infection is more frequent than supposed, finding as they do tubercular changes present in the great majority of placentas taken from tuberculous mothers. If a single focus heals, but leaves behind it a heightened sensitiveness for tuberculosis, then placental infection can play as important a rôle in the pathogenesis of tuberculosis as can intestinal infection. These ideas are, however, nothing but plausible theories.

We have seen how important the welfare of the epithelial coverings is for pathogenesis in general and especially for that of the infant. We also learned, in speaking of infectious diseases, that beside the entrance of the injurious particle, the so-called disposition of the patient is most important in the genesis of the disease. In other words, it is necessary for the invading substances, usually bacilli and their poisons, to meet cells capable of reacting. When this fails, disease will not appear. Under these conditions a natural immunity, as we say, against the invading disease is present. When bacilli enter an organism and produce a reaction, expressed by a certain disease, the symptom-complex characteristic of this disease will take a certain course.

The sum total of the symptoms, which are nothing more than the result of the reaction between the exciters and their products on the one hand and the human cells with their products on the other, represent to us the course of the disease. Its study, Pathology, will in its narrow sense occupy us later on.

The etiological factor of many diseases is at the present time known. For many others, this factor is merely a hypothetical one, worked out from cause and effect. The idea of an organic cause for the effect has never been proved. However, in explaining such diseases, we must accept at present the possibility of such a cause existing.

Rachitis.—To this latter group of diseases belongs one of the most important diseases of childhood, rachitis. The present idea, in contrast to that of older times, is that this disease is an affection of the entire organism, instead of the joints alone. We do not know the exciting cause necessary for the appearance of rachitis. We only know that the origination of rachitis, as well as the severity of its course, is favored by circumstances which have a general influence on the course of every disease. Nobody has been able to offer a satisfactory explanation for its cause or origin; even the most careful and thorough investigations of Pfaundler on the deposit of calcium salts in the animal tissues have proved of no avail.

It is most probable that rachitis is not a disease of the bones alone, but an infection of the entire organism, an idea which is persistently held at the present time. It appears to be a general disease, localized at the juncture of the cartilage and bone as well as at the epiphysis. This is explained by the fact that at these places the greatest activity and proliferation of cells take place. In other words, they are the places where the life of the bone is most active. The growth of every organ in the child goes on symmetrically, while that of the bones appears centralized. This growth does not take place throughout the entire bone, but seems to occur in certain peculiar zones, the cells of which have an especial power of development. The fact that this affection picks out these spots especially, finds its analogy in the fact that syphilis seems to choose with predilection these same places as well, while in the older child and the adult such is not the case. Rachitis depends therefore on the peculiar predisposition of the bones, and also on the apparently increased susceptibility at this point of most vigorous growth.

Rachitis occurs in Germany apparently endemically. The child otherwise absolutely sound, who has been fed on the milk of a healthy mother under the most hygienic conditions, is as little immune against rachitis as is the artificially nourished baby of the lower classes. The course of rachitis, however, will be found to be usually a light one in the former, and in the latter often severe. Kassowitz also emphasizes the importance of unfavorable hygienic environments in the appearance of rachitis, calling it "respiratory poison."

The Importance of Social Conditions.—This is a good time to show the importance of the social status (*i.e.*, the circumstances of the parents) in the appearance of diseases amongst infants.

Not only is poverty, with its social and unhygienic consequences, a great factor in disease of the adult, but also is it of the greatest importance to the child, especially the infant. The best environment for man consists of pure air, even temperature, sufficient light, and cleanliness. These are the most favorable hygienic conditions and are the essential factors for the maintenance of health in children. In view of this, it is not to be wondered that poor children show a much greater morbidity and mortality than do those in better circumstances. Insufficient nourishment is another harmful influence which predisposes children of the poor to disease.

Overfeeding.—On the other hand, we see that overfeeding may be harmful by producing an aphysiological stimulus. It is, however, a source of danger which attacks chiefly the children of the well-to-do. The work of Czerny must be looked upon as of great service in proving the injurious results of overfeeding. This is very common at present, with the exaggerated and over anxious care of children among the well-to-do, and can scarcely be too strongly condemned.

Diseases of Infancy.—The difference between the infant and the adult has been shown; also the distinction between the pathogenesis of infancy and that of later years. In the second year of life, when the child begins to crawl about and speak and when its diet approaches that of the adult, the pathogenesis will be seen to depend, not so much on the internal as on the external influences. These external conditions are many. Escherich has referred to the importance of filth infection ("Schmierinfection") in the pathogenesis during the period of first dentition. The importance of this dirt infection is enormous, in the genesis not only of tuberculosis but of all other forms of infectious disease,—stomatitis, angina, diphtheria, whooping-cough, and intestinal parasites. Escherich's idea is, that the growing curiosity of the teething infant, with its ability to crawl around and put dirty fingers into its mouth, has produced another group of diseases. This group is called by Feer "Schmutinfection," but Escherich prefers the name "Schmierinfection," thinking it is a better way to explain the method of transmission. The factor of "Schmierinfection" must be taken into consideration, with the increased opportunity for contagium at this time of life, because children, at least those of the poor, are thrown together very intimately on the streets, at the play-grounds, etc., and even sleep together in the same bed.

We should like to point out at this time another cause which may be of importance in the genesis of tuberculosis. This depends on the fact that tubercle bacilli may often be inhaled. The small child who has its mouth and nose closer to the floor dwells, so to speak, in an atmosphere which is much more laden with dust and tubercle bacilli than is that normally breathed by the adult, three feet higher. The chance for tubercular infection being the greatest between the first and second years of life, it naturally follows that the frequency of tuberculosis, as investigations have shown, suddenly increases between the years of two and four.

Tuberculosis is seen to exist in adults most frequently in the form of phthisis, but in childhood it tends to localize in the lymphatic system. This fact has not been well explained as yet. One imagines that it depends on the peculiar susceptibility and power of reaction of the lymph-nodes. What we have considered as scrofula these many years is often seen to develop into tuberculosis. There are many authors, however, who maintain that there is a non-tubercular form of scrofula. This seems to us no longer tenable, in view of the experience of Heubner, who declares in the last edition of his manual that all the cases of scrofula examined by him gave a positive test to tuberculin.

Diseases of Childhood.—During the fifth and sixth years of life the peculiarities of children approach more closely those of the adult. Pathogenesis, dependent on the life of the child, at this time in school,

now limits itself almost entirely to external influences. Nervous disorders appear as a result of mental work, scoliosis of crooked sitting positions, and myopia from reading and writing in insufficient light. The increased social intercourse of this period presents a good opportunity for the transmission of infectious diseases, and the mutual mental intercourse and psychical influences offer an opportunity for the appearance of certain nervous hysterical diseases. Of course, the proper predisposition must be present. In other respects, pathogenesis approaches that of the adult. Escherich shows how the crowded busy existence of to-day predisposes to certain traumatic diseases,—the increasing occurrence of appendicitis, etc. Tuberculosis, especially that of the lymph-nodes, is less often seen, the disease in general approaching that of the adult. Certain new and dangerous diseases of infectious origin are seen to appear at this time,—acute rheumatism, endocarditis, and chorea. The diseases at this period differ from the same in later life only in their course, just as we found so many did in the periods of first dentition and school life. The diseases appearing at this time (adolescence) are largely found among young girls. These are commonly chlorosis,—of the etiology and pathogenesis of which little is known to-day,—and certain hysterical diseases. Two diseases belong to this period of adolescence, which usually appear for the first time in the fifth decade of life. It is believed that they only appear after prolonged and persistent damage has been done, practically never if the etiological factors have been working only a short time. These two diseases are arteriosclerosis and gout. Diabetes is considered to occur only under the same conditions and so is found to be rare among children. The extraordinary rarity of malignant tumors in children is hard to explain, and can only be discussed on theoretical grounds without any practical or experimental evidence.

So far we have tried to explain the manner of origin of the most important diseases of childhood. Perhaps it would be better to consider how they spread. We still have the other question to answer; namely, How does the *course* of disease differ in childhood from that seen in adult life?

The Course of Disease in Childhood.—We have shown how most diseases arise from bacteria. From what has been said we know that disease is the expression of the reaction of the human being to the exciting cause of disease. Practical experience has taught us that these diseases do not appear immediately, but follow the supposed inoculation of the organism. Hours, days, and in some cases even weeks go by before the clinical symptoms appear. This interval, which extends from the time of infection to the appearance of the first symptoms, is known as the “incubation period.” This period of incubation, more or less long, which appears in each infectious disease, is explained by the

need of the specific or exciting cause of the disease when in the body, for a certain time in which to develop so that the toxins may be sufficient in amount to produce symptoms (a reaction). An initial resistance belonging to the organism must first be overcome, in order that the exciting cause may be felt and produce a demonstrable reaction in the form of a disease. This hypothesis may be plausible, but it is not satisfactory when considered in a critical light.

The theories and experimental investigations of von Pirquet and Schick are of much service in the question under consideration. They demonstrate clearly how the symptoms following the injection of diphtheritic serum, scarlet fever serum, and even the pure horse serum, which they designate as the "serum disease," may be able to give us new fundamental ideas on the incubation period as well on the course of disease. These experiments show that when the organism has had time to manufacture specific antibodies for the especial acute infectious disease attacking it, the disease appears. They claim that the disease, shown by the appearance of symptoms, is nothing more or less than the reaction between the toxins and the antibodies. The time preceding this, after infection has occurred, during which the organism forms the antibodies, is the period of incubation. Von Pirquet and Schick claim it is the same for measles as it is for the serum exanthem; namely, that the first symptom is developed at the time when the organism has formed antibodies to resist the invading cause. In other words, it is the time when the antibodies, appearing free in the blood, react with the circulating toxins of the disease, *i.e.*, of measles. The formation of antibodies appears, in this light, to be the signal for the appearance of the disease.

While this conception at the start seems to be improbable and paradoxical, that the body is sick because it is fighting against the toxins and is producing substances which we have considered formerly were only of use to the body, yet it strikes one as quite probable and a very logical idea when looked at from a critical point of view.

We should like in this connection to draw attention to a discovery of Jehle, which has seldom been noticed. He found that in acute pneumonia during childhood great numbers of agglutinins were formed, during the first days of the disease; they did not increase, however, but remained about the same, and during the crisis fell quickly. In the theory of von Pirquet and Schick, the agglutinins, being a rough estimate of the amount of the antibodies present, were thought to increase in number at the time of the crisis, quite the opposite from the above findings of Jehle.

The formation of antibodies is peculiarly essential for the body. The affinity or disposition in man to react to the presence of the exciting cause of pneumonia, measles, etc., compels him to produce anti-

bodies. The reactions between these and the toxins is seen in the appearance of the disease. The severity and duration of an illness depends on certain peculiarities in the course of the reaction (the intensity and duration).

The investigations of von Pirquet and Schick, most fundamental in the study of general pathology, are not the only ones supporting this view. Bail gives quite a number of experiments on animals, made by himself and his assistant. Infectious disease was experimentally produced and symptoms quite similar to the above were seen. Bail also asserts that the formation of immune bodies is inevitable in animals.

The tuberculin reaction, which shows an especial susceptibility of the tubercular patient for certain poisons of the tubercle bacilli, finds a clear explanation in the theories of von Pirquet and Schick. A patient ill with tuberculosis who, at the time, is in an afebrile period, reacts on the injection of tuberculin by a rise of temperature. This appears because in the patient's blood antibodies exist which immediately react with the injected tubercular toxins.

As we have seen, the course of a disease depends on the intensity and duration of the reaction, which takes place between the immune bodies and the tissues. This produces a symptom-complex which we generally speak of as a tuberculin reaction. As long as the toxins are manufactured and the exciting causes find favorable conditions for life in the organism, just so long will the disease exist. As soon as the cells essential for life stop acting, being damaged by the toxins, death appears. When the poisons are destroyed and no fresh ones are formed because the bacteria are dead, recovery takes place. This recovery, however, only occurs if the organism has not been weakened too much by the disease, which often leads on to slow decline and even to death. This happens when the cells are so damaged that they are unable to recover from the harm done by the toxins, as is illustrated in heart failure following diphtheria.

It is certainly true that recovery takes place when the cause of the disease has been removed. Injuries dependent on a disease often cannot be completely recovered from or healed, although the etiological factors have disappeared—i.e., when the factors have been at work for a long time. We refer to the advanced cases of atrophy, impossible of a complete cure, as the damage done to the intestine by improper feeding and other factors cannot be repaired.

The course of each disease depends on many factors. The prognosis is better when the number of untoward influences accompanying a disease is small. Apart from the injuries which as complications influence the course of disease, each disease has a typical course. This is, within certain limits, the same in children and adults. Some diseases are recognized to have a fairly definite characteristic course in child-

hood. We see that tuberculous adenitis predominates in children, as well as general miliary tuberculosis and tuberculous meningitis. The prognosis of tuberculous disease is graver during childhood than in later life.

All attempts to explain these things are mere conjectures. It is nothing more than a paraphrase to say that the lymphatic system of a child has a special susceptibility for tuberculosis.

Tuberculosis, while having a bad outlook during childhood, has an especially poor prognosis in the nursing infant. So-called inherited syphilis also has a very bad outlook. At this time of life measles, whooping-cough, and scarlet fever are prognostically bad. At this period resistance to all diseases is much weaker than among adults. It is hard to explain on what these facts depend. Perhaps it is that the child, who, as we know, grows most in the first years of life, has as a result of this expenditure only a small amount of resistance left for the invading bacteria.

While the course of so many diseases is more severe in the early years of life, the later years of childhood are seen to offer a more favorable outcome. Pneumonia, which is so often seen at this time, has a much better prognosis than when it occurs in adults. As we know well, an almost perfect prognosis is given in a child under 2 years, sick with acute lobar pneumonia.

The strength of the heart is of the greatest importance in the prognosis. Since valvular disease and the causes of fatty degeneration, alcohol, tobacco, and obesity, are all absent in children, it is perfectly understandable why the course of pneumonia in a child and in an adult may be so different.

While the uncomplicated course of every disease is a typical one, as a rule it is markedly influenced by complications. Factors which increase the source of infection give a poor prognosis. Previously existing diseases also have a bad influence on the prognosis. Tuberculosis previously existing can well be imagined to have a very bad effect on any acute infection. Unhygienic surroundings, such as poverty, deficient care, foul and dusty air, lack of light, and inadequate nourishment are factors having an unfavorable effect upon the course of the disease. Thus we can see that not only more diseases but severer types are seen among the poorer classes, when compared with the well-to-do. This influence on the morbidity and mortality of childhood, of unhygienic, unfavorable, surroundings due to poverty, is enormous.

SYMPTOMATOLOGY OF CHILDREN'S DISEASES

BY
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INTRODUCTION

THERE are two ways of presenting the relation between diseases and their symptoms. Either the symptoms peculiar to each disease may be enumerated, or the pathological conditions underlying the disease may be deduced from the several symptoms. The former method is commonly employed in the text books, while the latter, the semiotic system, is less often used.

The text book method is particularly serviceable for the study of the different types of disease and their character, while the semiotic method is more especially adapted to the needs of practical diagnosis. The physician when called to the sick bed observes or detects certain symptoms, and from these he arrives at his diagnosis, thus following a course directly opposite to that laid out in the text books. Herein lies the practical value of this method, which has been often adopted in monographs and essays but not in text books on the diseases of children. The "Symptomatology and Diagnosis of Children's Diseases" by Filatow (of Moscow) is an exception. This is an excellent work, but owing to the early death of the author it is now no longer in the first rank. I believe, therefore, that the following semiotic summary will meet an urgent practical need.

In many cases where an erroneous or negative diagnosis has been made, the mistake has been that the question as to the actual malady has not been considered. Certain symptoms have been detected at the time, which were perhaps purely accidental and did not call up the true picture of the disease. These symptoms, moreover, may mislead one and cause him to lose sight of the true state of affairs. The function of symptomatology is to check this tendency as far as possible.

What it cannot and must not do is to become a sort of automatic index to the diagnosis itself. It can never become a "key" to the diagnosis in any particular case. Such "keys" may be useful in recognizing objects of natural history, but they can never become the tools of the practising physician. The careful mental analysis in making a correct diagnosis can never be transformed into an automatic, subconscious process. Even the simplest mental picture of a disease can no more be

resolved into the sum of several symptoms than can a portrait into the successive strokes of the artist's brush. The result in either case would be a caricature.

It may seem unnecessary to emphasize the fact that a scientific diagnosis can be obtained only by a knowledge of the disease as a whole, including its various typical modifications of form and the characteristics of its stages. To obtain this knowledge there is but one text book, the clinical, which must be studied in person at the bedside. Whoever hopes to diagnose correctly by means of symptomatology without having carefully trained himself in the observation of children's diseases, will be speedily disillusioned.

A kindred danger lies in wait for the symptomatologist, and the following warning may place me beyond reproach: The value of single symptoms as such should never be exaggerated. The man who degenerates into a symptom monger will forfeit all capacity for wider views, and will stand helpless when the system he has carefully constructed leaves him in the lurch.

Everyone who constructs a practical symptomatology is unfortunately forced to frame some theory, and the high value of the method from a didactic point of view is all that reconciles me to the inevitable evil of such theories. I have found in clinical teaching that moderate theoretical instruction in symptomatology, in connection with the clinical course, increases the interest of the students and the success of the teaching. The clinical teacher should start from the obvious and more easily detected symptoms of the case in hand, to reach the diagnosis which he has in view. The students are more apt to feel the force of such an exposition than that of a theoretical discourse, which, moreover, they imagine could be replaced by reading. For this reason, it has been my practice to place my teaching and my case demonstrations almost wholly on a semiotic basis. The notes which I have accumulated for such purposes have furnished the ground-work for the present treatise. As the experience of a single writer can seldom, if ever, be the exclusive source for such an exposition, I have been obliged to draw additional data freely from many text books and other medical literature. I fully realize the danger thus entailed, of producing a pedantic work unfitted for the practitioner.

A concise manual of specific symptomatology can be allowed only a small space in a work like this, and I fear that that space has been exceeded. For this reason, I have been obliged in writing to employ a style as compressed and uninteresting as that of a lapidarist. I regard it solely as a means of reference, and to facilitate its use as such I call particular attention to the Index at the end of the section. This will be helpful in finding the different symptoms and divisions. It has been impossible in enumerating the diseases which give rise to numerous

symptoms even to attempt such a degree of completeness as the consultation of all the latest treatises would insure. Amid such a mass of details the general outline would be lost, and we could not see the forest for the trees. This consideration and the effort to omit nothing of real value, have placed me in many a painful dilemma. How nearly I have struck the golden mean must be determined by my younger colleagues in active practice, to whom this chapter is dedicated. With this idea in view, I feel myself entitled to deny the competence of any other tribunal, and above all that of the official theorists in this department, who will assuredly find much or all of my work inadequate. For example, the very grouping in the different divisions is in the highest degree unscientific. This must indeed be so, since this grouping can take account only of the purely external characteristics of a disease, such as a physician would easily detect, and not of those which lie deep in the inmost nature.

The possibility of such a misapprehension of my design as a whole, and of the detection of many other deficiencies of which I am not unconscious, cause me to look forward with a certain dread to the verdict of those of my own specialty. In this verdict due allowance must be made for the utter absence of modern models in my undertaking.

The arrangement of the Symptomatology is as follows: The title of each division, when it denotes a symptom or a syndrome is followed, when necessary by:

1. An elucidation of the wording and of the idea.
2. A brief description of the symptom, if it is evident, or directions for discovering and distinguishing it, if it is not apparent.
3. Data concerning the respective physiological conditions at different ages.
4. Predispositions in certain cases, whereby a symptom may be simulated.
5. An enumeration of the pathological conditions which may, but do not necessarily, accompany or produce the symptom under consideration. If, for example, under "Diazo Reaction in the Urine" croupous pneumonia is mentioned among the conditions, it must not be assumed that croupous pneumonia is always accompanied by diazo reaction; but only that in case a positive diazo reaction is present, pneumonia must be considered in the differential diagnosis. Single pathognomonic symptoms are practically unknown, but the few essential symptoms from whose absence definite conclusions might be drawn are so designated.

The pathological conditions in company with which the symptom named in the heading appears, are not in every case its actual cause; indeed, the connection may be simply coördinate, or even some relationship not yet understood.

To many of the diseases enumerated I have added, briefly:

1. Some other indications, whose presence may serve effectually to distinguish the respective diseases from other conditions mentioned close by.

2. Some etiological references, but only such as appear valuable for diagnosis.

Rare diseases which are the occasional cause of the symptoms named in the headings are put in brackets.

Roman figures after the names of disease (*e.g.*, meningitis tuberculosa I) indicate the stage of the disease.

Certain infectious diseases (such as the plague, typhus fever, Asiatic cholera, yellow fever, etc.) which seldom or never appear in our country, have been entirely omitted. This also is true of ailments which usually require the services of specialists, in the narrow sense of the term. I refer to certain surgical, orthopædical, otological, laryngological, psychiatric, and dermatological diseases. Many details which have little practical value are omitted, such as the determination of the different types of congenital chorea, local devices of the neurologists, etc. There is no discussion of symptoms which can be used only within narrow limits in children, or which do not differ materially from those in adults (*e.g.*, pneumothorax, pulmonary cavities). Symptoms having little value on account of their great frequency, such as "restlessness" and "fever," are not considered. Symptoms which demand special skill and require expensive bacteriological or chemical apparatus are not included.

1. FEVER WITHOUT DISTINCT, OR WITH EASILY OVER-LOOKED, PHYSICAL SIGNS

The temperature in children is to be taken in the rectum. The thermometer should be carefully cleaned, lubricated with vaseline, placed about an inch in the rectum, and allowed to remain 3 minutes. The mercury should always be shaken down immediately before taking the observation.

A. ABRUPT (SUDDEN) ONSET. ACCIDENTAL.

1. *Acute Infections:*

(a) During the period of incubation (measles, etc.).

(b) In the beginning and course of the following:

Typhoid (Temperature gradually rises, is then continuous, with daily variations from 1°-2° F. ($\frac{1}{2}$ °-1° C.); easily influenced by drugs. Typhoid state; generally no vomiting; leukopenia; after the fifth day enlarged spleen, roseola, Gruber-Widal reaction—all other signs may fail in infants).

A. ABRUPT (SUDDEN) ONSET. ACCIDENTAL.—Continued

Lobar pneumonia (central) } (Sudden rise, then continuous; frequently "meningismus";
 Acute osteomyelitis } herpes; leucocytosis).

Acute miliary tuberculosis (generally an irregular intermittent or remittent style) (Typhoid state, prostration; tachycardia, dyspnœa, cyanosis; tubercles in the choroid, thoracic hyperæsthesia).

Septicæmia } (High intermittent fever with chills; tachycardia, gallop
 Septic endocarditis } rhythm.)

Influenza (High, generally remittent fever, coryza, cough, bronchial râles).

Malaria (Regularly intermittent, cyclical).

Glandular fever (Pfeiffer) (Often intermittent; sore throat, congested tonsils, enlarged glands).

Diphtheria (incipient).

Acute polyarthritis.

2. Inflammatory Local Affections of Different Organs:

Otitis media and interna (Tenderness on moving the ear and by pressure on the mastoid process; clutching the head in infants).

Acute pharyngitis (Slight evidence of stenosis of throat and nose). Acute lymphadenitis after infections, septicæmia, typhoid, scarlatina, etc.

Acute endocarditis.

Pleurisy with effusion, especially in infants and in cases with insidious onset. Tuberculous meningitis (During prodromal period and beginning of brain irritation, slight fever; but high, intermittent fever in the suppurative form in infants).

Encephalitis and brain abscess (Changes in the pulse, "meningismus").

3. Toxic Conditions:

"Serum disease" (generally 13 days after the injection).

Intestinal autointoxication (this includes most of the cases of "febricula" and "ephemeral fever") (Fever of short duration, herpes and intestinal symptoms; improvement after catharsis, acetonuria).

4. Heat Congestion due to Insufficient Heat Radiation (In weak and premature infants in an incubator; heat prostration in older children).

NOTE.—Acute nephritis, pyelitis, cystitis, etc., may account for a doubtful fever, especially in young children. The urine should be carefully examined.

B. FREQUENTLY RECURRING FEVER (in the course of weeks and months).

1. Infectious Processes:

- (a) Malaria (Regularity of fever, blood findings, enlarged spleen, effect of quinine).
- (b) Septic ulcerative endocarditis (chronic form) (Irregular fever with tachycardia, prostration, rheumatic pains, formation of infarcts, heart murmurs later on).
- (c) Umbilical infections.
- (d) Tuberculosis (Generally evening exacerbations, marked rise and fall, thirst, skin temperature increased, sweats, absence of subjective signs of fever); also latent affection (a) of the bones and joints; (b) of the bronchial and mesentery lymph-glands; (c) of the lungs and pleura; (d) of the pericardium and peritoneum; (e) subacute and chronic miliary tuberculosis.

2. Anæmic Conditions:

Pernicious anæmia, splenic anæmia; pseudoleukæmia and leukæmia; infantile scurvy; "growing fever"; internal cephalæmatoma.

3. Hysteria (Thermoneuroses).

4. Chronic Arthritis.

2. SUBNORMAL TEMPERATURE

Erroneous conclusions may result from too short observation, dampness of axilla, or faulty position of the thermometer.

A. IN PREMATURE AND NEWBORN INFANTS.

Through radiation, severe disease conditions, bowel affections, sclerema, hæmorrhages, icterus gravis, tetanus, etc.

B. IN OLDER CHILDREN.

1. Accidental:

- (a) All forms of collapse, as in cholera infantum, typhoid, intussusception, appendicitis, dysentery, and after hæmorrhages.
- (b) After high temperatures in the intermission, especially crisis of infectious processes. Pneumonia, chronic lung infiltration, tuberculous peritonitis.
- (c) Severe acute intoxications (alcohol, etc.), paroxysmal hæmoglobinuria.
- (d) Cerebral diseases, cerebral and meningeal hæmorrhages, tuberculous meningitis, cortical epilepsy.

2. Habitual:

- (a) Starvation, œsophageal and pyloric stenoses, athrepsia.

B. IN OLDER CHILDREN.—Continued

- (b) Certain diseases of metabolism, diabetes mellitus and insipidus, myxœdema, Addison's disease.
- (c) Congenital and acquired heart diseases.

NOTE.—The surface temperature of the extremities is low in all forms of heart weakness, in anæmia after hæmorrhages, and in paralyzed limbs.

3. CHILLS

Generally before a rapid rise of temperature in older children or convulsions in young infants.

1. Acute General Infections:

Pneumonia, malaria (not the rule in young infants), cerebrospinal meningitis, ulcerative endocarditis, sepsis (with sinus thrombosis), pyæmia (with formation of infarcts), scarlet fever, mumps, erysipelas, acute anterior poliomyelitis, infectious jaundice.

2. Formation of Abscesses and Pus in Different Organs:

Abscess of the lungs, brain, kidneys; purulent meningitis, pericarditis, pleurisy, pyelocystitis, osteomyelitis.

3. Other Conditions:

Paroxysmal hæmoglobinuria; helminthiasis (?).

4. HYPERPYREXIA

Many neuropathic and other children have very high fever on slight causes. Temperatures of 106° F. (41° C.) and over are regarded as hyperpyrexia.

1. Acute Infections:

Especially those with preference for the nervous system: trismus and tetanus, cerebrospinal meningitis, etc.

2. Organic Cerebral Diseases:

Cerebral and meningeal hæmorrhage, venous hyperæmia of the brain, acute encephalitis, etc.

3. Local Inflammatory Processes in Other Organs:

Suppurative otitis media, suppurative pleurisy, pulmonary gangrene.

4. Sunstroke.**5. Hysteria.****5. POSITION AND POSTURE OF THE BODY IN DISEASE**

1. Passive dorsal posture (Stiff, immovable; superficial breathing; legs drawn up).—Found in many acute and chronic diseases, as pneumonia, typhoid, peritonitis, etc.

2. Abdominal posture.—Painful conditions of the back (abscesses, Pott's disease), photophobia.

3. Lateral posture (habit).—Pleurisy, pneumonia (at first on the healthy, later on the diseased side).

4. Gun Hammer posture (position "*en chien de fusil*"—lateral position with legs drawn up and head retracted).—Meningitis; chronic hydrocephalus.

5. Sitting posture.—In stenotic and cardiac dyspnoea, "*orthopnoea*"—see No. 74 (Restlessness, gesticulation, gripping at neck and chest, perspiration on face, dilated nostrils, etc.).

6. Sitting posture in Turkish fashion.—Rachitis in children from 2 to 4 years of age; spondylitis.

7. Uterine posture (similar to that of *fœtus in utero*).—Physiological in the newborn.

8. Abnormal positions found in spastic and flaccid paralyses, contractures, etc., *opisthotonus*, etc.

6. CONSTITUTIONAL TENDENCIES WITHOUT DISTINCT ORGANIC CHANGE IN OLDER CHILDREN

1. Tuberculosis.—Especially in chronic miliary tuberculosis, prodromal stage of tuberculous meningitis, bronchial and mesenteric tuberculosis, tuberculous peritonitis.

2. Pernicious and splenic anæmia.

3. Subacute gastro-intestinal catarrh; dysentery.

4. Helminthiasis.

5. Diabetes mellitus.

6. Chronic nephritis.

7. Masturbation.

8. Iodism and bromism.

7. INFANTILE ATROPHY

Emaciation, dried up "*old man*" appearance, wasting, anæmia, micro-polyadenitis; often hypertonia of the muscles, enlarged liver and spleen, subnormal temperature.

1. Athrepsia (Parrot) (Severe long-continued digestive disturbances, vomiting, diarrhoea and constipation, large, soft abdomen, grayish yellow hue of skin, emaciation and drying out of skin; increases slowly up to an extreme grade).

2. Tuberculous cachexia, especially in tuberculosis of the lymph-nodes and chronic caseous pneumonia (Frequently without cough, with slight temperature or none, slight digestive disturbances, frequently great hunger, enlarged liver and spleen, polyadenitis, tuberculin reaction).

3. Cachexia after non-specific bronchopneumonia.

4. Syphilitic cachexia (Changes on the face, coryza, muddy yellow complexion, fissures of lips, alopecia, seborrhœa, enlarged spleen, liver and testicles, eruption; effect of specific medication).

5. Cachexia after non-specific chronic furunculosis.

6. Inanition in pyloric stenosis and underfeeding (Chronic vomiting, visible stomach peristalsis, pyloric tumor).

8. ABNORMAL BUILD OR APPEARANCE

1. *Dwarfism*.—Myxœdema, mongoloid, micromelia, rachitis (for differential diagnosis see Table, pages 30 and 31), cretinism, hereditary syphilis, chronic arthritis (osteomalacia).

2. *Infantilism* (Retarded development of the genital organs, delayed puberty, absence of secondary sexual characteristics, childishness of mind and action in later years):

(a) Hereditary taint (alcoholism, tuberculosis, syphilis).

(b) Diseases and functional disorders of the liver (cirrhoses), of the thyroid (myxœdema), of the testicles, of the hypophysis.

(c) Acquired and congenital valvular heart disease.

(d) Progressive paralysis.

3. *Obesity*.—Must be differentiated from myxœdema, elephantiasis, acromegaly, dropsy, muscular pseudohypertrophy, "arthritis," diseases of the thyroid, overfeeding, mental and physical inactivity.

9. FACIAL EXPRESSION IN DISEASE

1. *Facies Dolorosa*.—Expression of pain.

(a) Frequent, intermittent pain (Eyes tightly closed, contracted forehead, tectus, crying with wide open mouth, redness and sweating of face).—Colic; difficult dentition; dysuria.—Crying without long pauses, but with movements of all limbs: Otitis, etc.

(b) Continuous pain (Eyes open, mouth half-closed, eyebrows raised, mouth distorted, crying and whining, moanful sighing).—Pneumonia, pleurisy, peritonitis.

2. *Facies Luctuosa*.—Expression sad and woeful (Lower jaw and lips protruded and open, deep folds and furrows, forehead vertically wrinkled).—Tuberculosis and other chronic diseases.

3. *Facies Anxiosa*.—Expression anxious and fearful:

(a) Agitated, dyspnœa from stenosis (Nostrils dilated, cyanosis of lips, depression of suprasternal notch).—Pavor nocturnus.

(b) Rigid.—Heart disease, infectious myocarditis after scarlatina, diphtheria gravis.

4. *Facies Hydrocephalica*.—Expression earnest, thoughtful, and

DIFFERENTIAL DIAGNOSIS OF MYXEDEMA, MONGOLOID, MICROMELIA AND RACHITIS.

	Myxedema.	Mongoloid.	Micromelia, chondrodystrophy.	Rachitis.
Sex	Seldom in boys (10-20 per cent.).	Both sexes equally affected	Almost always in girls	Both sexes equally affected.
Time of onset	Congenital—observed in first weeks of life.	Congenital	Congenital	Second to fourth year of life.
Length of body	Decidedly shortened—more apparent in later years.	Markedly shortened	Very decidedly shortened	Slightly shortened.
Proportions of body	Nearly normal. Neck plump, skull large, often asymmetrical.	Nearly normal. Obesity	Decided disproportion. Skull very large, trunk normal, extremities very short (especially femur and humerus)—"Dachshund" type.	Nearly normal. Skull large, four-cornered; extremities plump, short.
Time of ossification, as seen in hand by radiogram.	Delayed.	Normal	Normal or premature	Delayed.
Calcification of long bones and union of epiphyses.	Delayed.	Normal	Premature	Delayed.
Size of epiphyses	Enlarged		May be very large	Enlarged.
Closure of fontanelle	Greatly delayed; edges of bone hard, sutures obliterated. (Never complicated with rachitis.)	Delayed; edges of bones soft; craniotabes; sutures open. May be complicated with rachitis.	Delayed; edges of bones hard	Delayed (fourth to sixth year), but not so long as in myxedema.
Dentition	Greatly delayed; teeth irregular and bad.	Delayed	Not delayed	Delayed; teeth irregular and bad.
Joints	Very loose; may be overdistended without pain.	Extremely loose	Often loose; frequently congenital dislocation of hip.	Loose.
Skin	Mucoid edema, doughy, gelatinous, yellow. Superficial layer dry, shiny; deep layers thickened.	Much adipose tissue, soft, shiny, smooth.	Smooth, soft; in folds on account of shortness of long bones.	Soft, tender.
Perspiration	Lessened	Normal	Normal	Increased.
Temperature	Often subnormal	Normal	Normal	Normal.
Expression	Apathetic or anxious (forehead wrinkled, face puffy and full).	Cheerful, comical, or stupid	Cretenoid	Not idiotic.

Color of face	Pale, yellow, earthy	Red as if painted	Pale
Hair	Sparse, thick, brittle; high forehead; no pubic hair. (Bad nails.)	Normal	Thin, soft; occiput often bald.
Eyelids	Thickened, pseudo-ecdematous; narrow space; epicanthus; blepharitis ciliaris.	Slit shape; epicanthus	Normal.
Bridge of nose	Depressed. Nose tumid; mucous membrane thickened.	Depressed, wide; nose small	Normal.
External ear	Frequently deformed	Frequently deformed	Normal.
Mouth	Wide; lips thick and prominent	Generally open; small; very wide on laughing.	Wide, on account of deformity of alveolar process.
Tongue	Enlarged. Dysphagia and salivation.	Protruding, but normal in size	Normal.
Palate	High-arched	High-arched	May be somewhat arched.
Bowels	Constipation, the result of bowel stony; frog belly; frequently prolapsus recti.	Constipated	Constipation; bowel atony.
Navel	Frequently umbilical hernia	Frequently umbilical hernia	Umbilical hernia frequent.
Intelligence	Backward; not malicious.	Backward	Normal.
Behavior	Stupid, sleepy	Restless	Restless; ill humor.
Walking	Delayed; great fear and unsteadiness when attempted.	Delayed	Backward; delayed.
Sexual instinct	Lessened; organs hypoplastic	Lessened	Normal.
Respiration	With stridor. Voice raw, deep	With stridor	Normal in absence of affections of respiratory system.
Complications	Disturbances of hearing and speaking.	Deformities, heart diseases	Deformities of osseous system.
Thyroid gland	Absent	May be absent	Normal.
Pathogenesis	Hypothyroidism	Anomalies of some function of the thyroid gland, or other glands.	Functional anomaly of "inner secretion" of some gland.
Thyroid therapy	Rapid improvement of all symptoms.	Improves constipation, hernia, dentition; generally without effect on grunting.	No specific effect.

resigned (Eyes brilliant, pupils contracted, forehead wrinkled, mouth closed, teeth clenched, alternating redness and pallor of cheeks).—Meningitis, especially tuberculous.

5. *Facies Paralytica*.—Expression helpless (Eyes lustreless, dull, lids drooping, cornea hazy, pupils dilated, often strabismus).—Terminal stage of conditions under 1, 3, and 4.

6. *Facies Sardonica*.—Expression morose, ironic (Forehead and cheeks wrinkled, eyelids slightly contracted, alæ of nose dilated, lips puckered and protruding, frequently frothing of mouth, masseters prominent).—Trismus and tetanus (Chorea). Changeable expression in tetany.

7. *Facies Fastidiosa*.—Expression of disgust, ennui, nausea (Mouth open, nasolabial folds prominent, nose "turned up," tongue protruding, lips extended outward, empty chewing).—Dyspepsia, gastritis, and other abdominal processes.

8. *Facies Senilis* ("mine de Voltaire").—Expression mummy-like (Pointed nose, sunken bulbs, eyes expressionless, immovable, skin deeply wrinkled).—Athrepsia, syphilis; chronic internal hydrocephalus.

9. *Facies Hippocratica seu Abdominalis*.—Expression cadaveric, empty (Border of eyes dark, leadish gray color of skin, cornea lustreless and covered with mucus, eyelids immovable and cyanotic, pinched nose).—Severe abdominal processes, cholera nostras, peritonitis, appendicitis, occasionally in other conditions with a sudden onset.

10. *Facies Libidinosa*.—Expression sensual (Eyes staring, flashing, lips firmly closed).—Masturbation.

11. *Facies Rigida* (mask face).—Expression one of astonishment (Countenance rigid, eyeballs immovable, eyelids wide open, eyebrows raised).—Cerebral affections, paraplegic paralysis, cerebral palsy, etc. Tetany, pseudotetanus, post-diphtheritic, meningitic, and other convulsions and paralytic forms. Progressive facial amyotrophy, progressive bulbar paralysis, myotonia, sclerœdema.

12. *Facies Idiotes*.—Expression stupid, empty (Mouth open, tongue protruding, lids half closed, forehead wrinkled).—Idiocy.

For facies in myxœdema, mongolism, micromegaly, see Table on pages 30 and 31.

13. *Facies of Inattention*.—Expression forlorn, weary, dull, suspicious (Low forehead, stupid look, tower-shaped skull, asymmetrical face, nose small, nostrils fixed, nasolabial fold obliterated, mouth open, lower jaw relaxed and protruding, cheeks flattened, defects of external ear, high palate), degeneration, adenoids.

14. *Facies Hutchinsonii* (Bilateral ptosis, wrinkled brow, eyebrows raised, nystagmus).—Progressive ophthalmoplegia.

<i>Facies Morbillosa,</i> <i>Facies Pertussea,</i> <i>Facies Scrophulosorum, etc.</i>	}	Described in detail elsewhere.
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NOTE.—Vein phenomena, *see* No. 90; Color of skin (erythema, cyanosis, etc.), *see* No. 182; (Edema, *see* No. 190; Lymph-node swellings, *see* No. 101; Paralysis, *see* No. 157.

10. CRYING IN VERY YOUNG CHILDREN

1. *Due to Some Particular Cause:*

- (a) Raising or handling the infant.—Infantile scurvy, acute rachitis.
- (b) Before and during bowel movements.—Constipation, fissure of anus.
- (c) Before and during micturition.—Dysuria, ischuria.
- (d) Before and during feeding.—Affections of upper respiratory and digestive tract, pyloric stenosis.
- (e) Entrance of physician.

2. *Spontaneous:*

- (a) Bodily discomfort.—Hunger, cold extremities, wet napkins, uncomfortable clothing, insect bites.
- (b) Bodily pain (Loud, prolonged cries with painful expression of face).
- (c) Colic (Sudden onset and termination, flatulence and meteorism, legs drawn up.—Pressure on abdomen gives relief).
- (d) Otitis media (Increased pain on pressure over external ear, also on swallowing and sucking).

Loud and severe crying will exclude such diseases as atelectasis of the lungs, advanced peritonitis, pleuropneumonia and croup.

Hydrocephalic cry (Generally at night, abrupt, without sufficient cause during sleep, shrill and piercing).—Meningitis, hydrocephalus, encephalitis, sinus thrombosis, before an epileptic attack. Rarely "meningismus" in acute infections.

(Aphonia, *see* No. 70).

11. CHANGES OF THE MOUTH AND LIPS

1. *Macrostomia*.—Wide open mouth.—Myxœdema, mongolism, rickets.

2. *Microstomia*.—Congenital; scar tissue.—Syphilis.

3. *Open Mouth:*

- (a) Habitual.—Nasal and pharyngeal stenoses (especially adenoids, chronic rhinitis in scrofula); idiocy.
- (b) Accidental.—Paralysis of trigeminus, stomatitis, inflammatory conditions of the throat.

4. *Enlarged Upper Lip*.—Scrofula (with eczema and ulceration); irritation of the skin from rhinitis; adenoids; syphilis; myxœdema.

5. *Fissured Lips, Ulceration, and Scars*.—Syphilis (radial grooves about the lips, border of lips not well defined); stomatitis of different kinds during and after typhoid, pneumonia, scarlet fever, measles,

dermatitis exfoliativa, tuberculosis, scrofula; also in febrile conditions, profuse diarrhœa, etc.

6. *Ulcer at Corner of Mouth*.—*Perlèche*, “*Faule Ecken*” (Generally symmetrical, extends from angle of mouth; no enlarged glands; surrounding skin red; contagious).

7. *Membrane on Lips*.—Diphtheria (thick membrane, later separates intact); stomatitis maculofibrinosa; typhoid; syphilis; burns and scalds.

8. *Staining of Lips*.—After taking colored food or drink. In chronic metallic poisoning (lead, etc.).

9. *Paralysis of Lips*.—*See Bulbar Paralysis, Paresis and Paralysis of the Facial Nerves.*

12. SWELLING IN THE REGION OF THE PAROTID GLAND

(Between mastoid process, condyloid process, and ear)

1. *Enlargement of Parotid Gland and Surrounding Connective Tissue* (Lower lobe of ear protrudes):

(a) *Parotitis epidemica* (Mumps) (With serous infiltration of surrounding tissue as far as the cheeks, eyelids, and throat; swelling doughy, gland itself harder; generally bilateral and simultaneous).

(b) *Parotitis concomitans*.—Diseases of mouth; otitis.

(c) *Parotitis metastatica* (slower course; frequently suppurative; painful, swelling smaller and harder; generally unilaterally).—Scarlet fever, measles, whooping-cough, sepsis neonatorum, influenza, diphtheria, typhoid, dysentery.

(d) *Parotitis toxica* (bilateral; rapid healing after discontinuance of poison).—Iodism.

(e) (Infiltration of parotid in chronic leukæmia.)

2. *Swelling of Neighboring Auricular Lymph-nodes* (Depression between the mastoid and condyloid processes remains unaffected):

(a) Lymphadenitis, preauricular } (*See Acute Inflammation of*
(b) Pfeiffer's glandular fever } *Lymphatic Glands.*)

13. DEFORMITIES AND ANOMALIES OF THE SKULL

Physiological.—The only fontanelle to be considered in the newly-born is the large fontanelle. Its long diameter is about 4 cm. (1½ in.) and the cross diameter is 2 to 3 cm. (¾ to 1½ in.). It grows smaller during the first year and by the twelfth to sixteenth month should be completely closed and ossified.

1. *Plagiocephaly* (Asymmetrical skull).—Constant lying on one side. One-side compression of skull *in utero*. Defect of one hemisphere, birth injuries, premature unilateral closure of sutures; brain tumors,

atrophy of brain; unilateral atrophy of face, torticollis. (Frequently associated with epilepsy, rachitis, infantile cerebral palsies).

2. *Acrocephaly, Pyrgocephaly* (Pointed and tower-shaped skull).—Stigma of degeneration, adenoid vegetations. (Associated with idiocy, exophthalmus, prognathos, atrophy of optic nerve.)

3. *Microcephaly* (Skull small, round, and short; forehead and occiput flat), associated with an "ape-like" countenance, protruding ears, prominent lower jaw, narrow orbits.—Idiocy, degeneration, hypoplasia, or after foetal diseases of the brain.

4. *Macrocephaly* in the widest sense:

(a) Rachitic deformity (Skull square, frontal and parietal tuberosities prominent. Craniotabes; large and pulsating fontanelle which does not bulge. Appears about the end of the first year; no pressure symptoms; no psychical disturbances. Circumference of head not much enlarged. Other rachitis symptoms).

(b) In Hypertrophica cerebri (Appears early; severe cerebral symptoms; forehead and occiput protrude, fontanelle flat and not tense).

(c) Hydrocephalus, chronic internal and external, congenital and acquired. (Skull round or pear-shaped, cranium evenly distended. Fontanelle bulging, tense, no pulsation, four-cornered with hard edges. Sutures open. No craniotabes. Frequently cerebral pressure symptoms, spasms, contractures, rigidity; abnormal prominence of the eyes, exophthalmus, "hydrocephalic face," mental defects).—Congenital syphilis, meningitisluetica (cranial deformity appears generally in the first half-year, is not excessive; imbecility).—Rachitis, Organic cerebral disorders (tumors, encephalitis, lepto- and pachymeningitis, venous thrombosis in the stage of recovery).

5. *Saddle- or Cross-shaped Head* ("Tête carrée," caput natiforme) (Cranium as if pushed up from in front, forehead keel-shaped, all tuberosities prominent, depression of lambdoid suture):

(a) In the first month of life, with small fontanelle and hard bones.—Hereditary syphilis.

(b) In the second and third year, with large fontanelle and soft bones.—Rachitis.

6. *Flat-head* (Cranium wide, flat and low).—Condition of early developed cretinism.

7. *Flattened Occiput*.—Rachitis.

8. *Opened Sutures*.—Rachitis.

9. *Circumscribed Protrusions and Depressions*.—Tumors, encephaloceles, injuries.

10. "*Craniotabes, Congenital.*"—*Spina bifida*, chondrodystrophy, osteogenesis imperfecta. Injuries to skull (forceps).

11. *Craniotabes, Acquired.*—Rachitis (with loss of elasticity on the affected spots on the occiput, parchment-like sensation, observed in the first few months of life). Chronic hydrocephalus.

12. *Delayed Closure of Fontanelles.*—Rachitis, hydrocephalus, myxœdema, mongoloid, micromelia.

13. *Protrusion of the Fontanelle* (during crying is physiological), often with separation of cranial bones.—Increased intracranial pressure, hyperæmia, hæmorrhage, tumors of the brain and meninges. Hydrocephalus, acute (meningitis serosa and cerebrospinal) and chronic. Thrombosis of the longitudinal sinus; "meningismus" in infections (pneumonia, acute nephritis, etc.).

14. *Depression of the Fontanelle* (often with overlapping of cranial bones).—Lessened intracranial pressure. Loss of fluid (diarrhœa) atrophy, hydrocephaloid, weak heart, paralytic stage of meningitis.

15. *Swelling of the Head in the Newly-Born:*

(a) *Caput succedaneum* (Doughy consistence, skin œdematous, bluish discoloration, borders diffuse without relation to sutures, situated at the presenting part of the skull. Is present during birth and quickly disappears).

(b) *Cephalœmatoma*, true external (Fluctuating, skin normal, does not extend beyond borders of bone, non-pulsating and non-reducible. Generally appears from 2 to 3 days after birth and does not disappear for at least 6 months. Most frequent over right parietal bone).

(b*) *Cephalœmatoma*, false external (As above, only is not limited over one bone, disappears more quickly, skin discolored).

(c) Abscesses and periostitis (Fluctuation, pain, inflammation of the skin).

(d) *Hydromeningocele* and *encephalocele* (Situated over the sutures, generally over roof of nose or on the neck, medial, soft, elastic, fluctuating and pulsating, more prominent on forced expiration. Often reducible, causing cerebral symptoms).

[Congenital tumors (Lipoma, sarcoma, dermoid cysts, blood cysts).]

17. *Tumors of the Head in Older Children.*—Traumatic hæmatoma and abscess. Sarcoma and syphilis of the cranial bones.

14. MANIFEST CHANGES OF THE NECK

1. *Forced Lateral Flexion of the Head, Torticollis:*

(a) Cicatricial torticollis (Oblique position of the head to one side—the sound side—and downward. Passive turn-

1. *Forced Lateral Flexion of the Head, Torticollis:—Continued*

ing difficult. Is generally observed during the first few months of life. Cause nearly always abnormal mechanical difficulties at birth, and followed by facial scoliosis with atrophy of one-half of the face. Local lesions in muscle). Hæmatoma of sternomastoid.

- (b) Spastic torticollis (Turning the face to the healthy side, elevation of chin, sinking of ear on the affected side; there are always other spinal and cerebral signs. Muscles rigidly prominent, palpable). Spinal meningitis, tumors of the crura cerebri and corpora quadrigemina. Hysteria.
- (c) Paralytic torticollis (Oblique position of the head to the affected side and upward, passive turning to the well side easily accomplished).—Paralysis of accessorii.
- (d) Rheumatic torticollis (Spinal processes of the cervical vertebræ or cervical muscles very painful on pressure; fever, and other articular and muscular affections).—Spondylarthritis cervicalis and muscular rheumatism M. sternocleidomastoideus, trapezius, splenius).
- (e) Torticollis to relieve tension and pain caused by other inflammatory conditions in the neighborhood, in bones (Pott's disease), in glands (lymphadenitis, retropharyngeal abscess) in epidermis, and in mucous membranes (otitis).

2. *Deflection of the Cervical Vertebral Column:*

- (a) Cerebrospinal spasm (see No. 136), dyspnœa (see No. 74).
- (b) Spondylitis cervicalis. Retropharyngeal abscess. Myxœdema.

3. *"Tumors" in the Region of the Neck:*

(a) Of the thyroid gland:

- (a) Physiologically there is moderate enlargement during sleep, at puberty, after bodily exertion.
- (β) Swelling, due to hyperæmia (from mechanical causes and in acute infectious diseases: diphtheria, scarlet fever, measles, German measles, mumps, whooping-cough, typhoid fever) (Light stridor, reddening of the face, tachycardia, no really inflammatory manifestations).
- (γ) Thyroiditis (Head bent forward, sternomastoid pressed back, with tension over the tumor; hard and painful. In acute thyroiditis generally suppuration, compression of nerves, vessels, and trachea).—Trauma, acute infectious diseases, as above, also polyarthritis and mumps.
- (δ) [Tuberculosis and syphilis, also true tumors of the thyroid gland.]

3. "*Tumors*" in the *Region of the Neck*:—*Continued*

- (e) Goitre, acquired, frequently occurring at puberty; congenital, endemic.
- (f) Exophthalmic goitre (Tumor soft, vascular, compressible, pulsating, often growing rapidly, in general only moderately large, and other typical manifestations. Very rare before puberty).
- (g) Endemic cretinism (Goitre, generally occurring only at the age of 2 or 5 years, often more developed on the right than on the left side; hereditary).
- (b) Of the lymphatic and salivary glands:
 - (a) Acute lymphadenitis and perilymphadenitis in severe infectious pharyngeal diseases ("cou proconsulaire" in diphtheria, scarlet fever, mumps, etc.). Chronic lymphadenitis in scrofulosis, tuberculosis, pseudo-leukæmia, eczemas, etc. (*see No. 101 and following*).
- (c) Of the sternocleidomastoid:
 - (a) Hæmatoma (Only during the first 2 to 3 weeks of life, a tangible hard nodule the size of a hazel-nut in the middle of the muscle or at the sternal insertion, more often right than left).
 - (β) [Cystic hygroma of the neck (Congenital, multilocular, cystic tumors between lower jaw and clavicle, growing rapidly, tense, filling the entire soft parts of the neck).]
 - (γ) [Blood cysts of the neck (as above, but less tense, communicating with veins, filled with blood).]

4. *Fistulas of the Neck*:

- (a) [Congenital median fistula (exterior opening in the median line; duct blind or communicating with the trachea).]
- (b) Congenital lateral fistula (exterior opening generally near the beginning of the sternomastoid; duct small, either blind or communicating with pharynx, discharging tenacious mucus).
- (c) Acquired fistulas following glandular suppuration in scrofulosis, etc.

15. DEFORMITIES OF THE THORAX

1. *Barrel Shape* (Increase in all diameters, especially the lateral and anteroposterior; upper aperture and ribs almost horizontal. Shape similar to that at the height of inspiration; epigastric angle larger, Louis' angle distinct; intercostal spaces obliterated).—Physiological in the newly-born (to a certain extent). Emphysema, bronchitis, pneumonia, whooping-cough; certain forms of croup, bronchiectasis, pneumothorax.

2. *Contracted Shape* (Thorax flat, narrow, long, intercostal spaces wide, ribs yielding, epigastric angle acute, Louis' angle distinct; pectoral arch protruding. Respiration shallow, chiefly abdominal).—Tuberculous diathesis. Stenosis of the upper air-tracts. (Congenital atresia of the nose, adenoid vegetations, chronic stenosis of the larynx, tracheal cicatrices with stricture, etc.).

3. *Funnel Shape* (Funnel-shaped depression of the lowest part of the sternum and of the last costal cartilage, notably in inspiration):

(a) Congenital malformation with degeneration (in this case enormous contracture).

(b) Rachitis.

(c) Infantile scurvy (with symmetrical costal depression).

4. *Pigeon Breast* (Flattening or lateral sinking between the fourth and eighth ribs; sternum prominent, costal arches turned up).—Rachitis (here also "Rosenkranz" or "rachitic rosary" (swellings at the junction of cartilages and ribs), Harrison's diaphragmatic groove level with the ensiform process, multiple costal inflections, flattening of the dorsal surface, frequent asymmetries). After severe whooping-cough, congenital heart disease. Sometimes in chronic stenosis of the upper air-passages (as above, Mongoloid).

5. *Asymmetric Shape*:

(a) Congenital asymmetry.

(b) Unilateral dilatation in pneumothorax, pleural effusion.

(c) Unilateral contraction in pleural adhesions, contracted lung (apices), scoliosis.

16. ENLARGEMENTS OF THE THORACIC WALL

1. *Swelling of the Mammary Glands*:

Physiological or normal: accumulation of secretion in the glands during first few days after birth; budding of female breast at beginning of puberty. [Physiological engorgement of male breast at puberty.]

(a) In newborn infants:

(a) Distention by milk (Beginning in the first few days after birth. Duration two to three weeks).

(β) Septic mastitis in sepsis neonatorum.

(γ) Abscesses and phlegmons of the gland.

(b) In older children:

(a) Mumps (mostly unilateral in both sexes).

(β) [True Tumors.]

2. *Suppurative Abscesses*:

(a) Spontaneous opening in empyema (generally below the nipple, sometimes lower).

(b) Abscesses in costal caries, suppuration of bronchial glands, sternal periostitis, peripleuritis.

3. *Obliteration and Bulging of Intercostal Spaces:*

(a) Emphysema, pleurisy with effusion (rare, especially in empyema), and pericarditis.

4. *Rachitic Rosary* (Double nodular swellings at the junction of the cartilages and ribs, especially the lower).—Rachitis, myxœdema. (To a slight extent physiological.)

5. *Hernia of the Lung* (Soft tumor in the clavicular region, size of a nut, feels like a feather cushion, respiration distinctly audible).

6. *Bulging of the Precordium:*

7. *Hypertrophy of the heart, pericarditis.*

17. CHANGES OF THE ABDOMINAL WALL

Obliteration and depression, *see No. 52* and following. Umbilical changes, *see No. 62.*

1. Striated hyperæmia from the umbilicus to both sides of the bladder.—Inflammation of umbilical arteries.

2. "Lymph-nodules" (varicose and congested lymph vessels) (Hard nodules, pinhead size, below the skin, especially in the umbilical region, palpable and visible, especially in emaciation).—Follicular enteritis, atrophy, tuberculosis of the mesenteric glands.

3. *Lineæ albicantes*.—After distention of the peritoneum.

4. *Hernia of the abdominal wall* (Bulging of the median portions, of Petit's or Lesshaft's triangle).—Increased abdominal pressure in congenital malformations and aplasia of the musculature, injuries, separation of the recti muscles, etc. Pseudohernia in paresis of the abdominal muscles. (*See No. 163.*)

5. *Caput medusæ*.—Thrombosis of abdominal veins, hepatitis (syphilitic), pressure of abdominal tumor on abdominal veins; any increased restriction of space in the abdomen (peritoneal tuberculosis). Cachexias of all kinds.

6. *Parietal exstrophy of the bladder* (Umbilicus and genitals normal; in the region of the bladder the mucous membrane of the bladder forms a flesh-red tumor with two little protruding secretory heads: openings of ureters).

7. *Total exstrophy of the bladder* (Tumor as above, penis short, total epispadias, umbilicus lowered into the tumor, separation of symphysis pubis).

18. CHANGES IN THE SACROLUMBAR REGION

(Congenital Tumors)

1. *Caput succedaneum* in breech delivery.

2. *Spinal meningocele* (Generally delicate pediculated, fluctuating, translucent tumors in the posterior median line, the compression of which tightens the fontanelle; is covered with normal skin; no umbilical

retraction, no other malformation, generally no fissures demonstrable at the spinal column and no paralysis; rare).

3. Spinal myelo-(meningo-) cele (Tumor as above, but more broadly pediculated, not transparent, reposition impossible; compression causes tension of fontanelle and nervous manifestations of irritation; the skin close to the tumor is thickened to a ridge and assumes an umbilical shape higher up. In the spinal column below the tumors, separations of vertebrae; the cranial bones frequently show lacunæ. Paralysis of the lower extremities and sphincters; frequent convulsions).

4. Myelocystocele (Broadly sessile, covered with normal skin; generally no paralysis).

5. Sacral tumors.—Lymphangiomata, lipomata (among which often spina bifida occulta), teratomata (X-ray examination to distinguish from spina bifida).

19. CHANGES IN THE HANDS AND FEET

1. *Claw Hand*.—Paralysis of ulnar, progressive muscular atrophy, certain affections of the spinal system.

2. *Drum-stick Fingers* (Clublike swelling of terminal members, with malformation of nails, and cyanosis; (without change of the bones):

- (a) Congenital heart disease (the only cause occurring in suckling infants).
- (b) Lingered coughs.—Pulmonary tuberculosis, bronchiectasis with considerable fetid expectoration, chronic bronchopneumonia and empyema, pericardial adhesions.
- (c) Infantile hepatic cirrhoses (among others, hereditary syphilis).

3. *Fusiform Swelling of Phalanges*:

- (a) Spina ventosa (Affects generally only one to two base, middle, or end phalanges; inflammatory manifestations; later hyperæmia, distortions and perforation of the skin; very rare in infants).—Tuberculosis.
- (b) Syphilitic osteitis (Mostly multiple, frequently in end phalanges, no ulceration, necrosis, or caries; sometimes hyperæmia of the skin; predominating in infants).
- (c) Rachitic and myxœdematous deformities (olive shape of phalanges separated by furrows; no change in soft parts).

4. *Dystrophic Ulcer Formation*, necrosis, panaritium, shedding of nails with cyanosis, scleroderma, sclerodactylia, syringomyelia.

5. *Hyperplasia of all Tissue Strata*.—Acromegaly (Enlargement of thymus, spleen and thyroid, affection of hypophyses). Elephantiasis after lymphangitis, erysipelas (frequently involving not only the feet,

but also scrotum and prépuce). Chronic arthritis (abnormal thickness and length of the great toe). Myxœdema (paw hand).

6. *Web Formation* in idiots.

7. *Other Malformations*.

8. *Erythromelalgia* and Raynaud's disease.

For abnormal position of hands and feet in spastic and other paralysis, exaggerated muscular tonus, contractures, ataxia, etc., *see* Nos. 132, 134, 157, and following.

20. CHANGES IN APPETITE

1. *Increased Desire to Eat, Avidity to Eat, Bulimia, Exaggerated Ingestion of Food:*

- (a) As a bad habit in the healthy.
- (b) Disturbed digestive function, intestinal irritation. Tuberculosis of mesenteric glands, athrepsia of suckling infants, pancreatic affections, helminthiasis.
- (c) After wasting diseases, especially typhoid, pneumonia (disappears after convalescence).
- (d) Idiocy. Cerebral affections (especially affections of hypophyses).
- (e) Diabetes mellitus, "arthritis," obesity.

2. *Decreased Desire to Eat, Loss of Appetite, Anorexia:*

- (a) Simulated by defective ability to imbibe, masticate, and swallow (*q.v.*). In breast-fed children through insufficiency of mother's milk, difficult flow, nipples difficult to hold. Also in pyloric stenosis with cardiac spasm; also in asphyxia and debility; through overestimation by relatives of nourishment required. Breast milk should be weighed and the artificial food given measured.
- (b) Irrational manner of living and faulty dietary regime. Overfeeding (albumin).
- (c) Anæmic conditions: "School anæmia," chlorosis, pernicious anæmia, leukæmia, pulmonary tuberculosis.
- (d) Acute febrile infections, especially influenza, typhoid, diphtheria, miliary tuberculosis, pneumonia, whooping-cough, cerebrospinal meningitis.
- (e) Acute and chronic gastro-intestinal affections, helminthiasis, tuberculous peritonitis and purulent appendicitis, gastric and intestinal atony.
- (f) Acute and chronic intoxications.
- (g) Genito-urinary affections of girls. Chronic vulvovaginitis and cystitis; uræmia.
- (h) Prodromal in tuberculous meningitis.
- (i) Hysteria.

3. *Parorexia, Geophagia, Coprophagia, Pica*.—Chlorosis and other anæmias, irritable debility of the nervous system, rachitis, idiocy.

21. PATHOLOGICAL INCREASE OF THIRST, POLYDIPSIA

1. Simulated by physiological thirst after ingestion of strongly salted or spiced dishes.
2. All acute febrile diseases, especially pneumonia, etc.
3. Gastro-intestinal conditions with or without diarrhœa; dyspepsia (with gastric atony), enteritis, colitis, dysentery, helminthiasis, appendicitis, peritonitis, pancreatic conditions.
4. Rachitis (perspiration and diarrhœa).
5. Affections of the hypophysis.
6. Incipient acute nephritis.
7. Intoxications: botulism, atropine, digitalis.
8. Primary polyuria, and tachyuria; diabetes mellitus, insipidus, etc. (*q.v.*).

22. CHANGES IN SALIVARY SECRETIONS

1. *Salivation* (pathologically increased):
 - (a) Simulated by the flow of (non-increased) saliva in various derangements of innervation; poli-encephalitis, facial paralysis, infantile karyolysis, psychoses, idiocy; macroglossia; in the beginning of the secretion.
 - (b) All inflammatory processes in mouth and fauces (scarlatina, diphtheria, mumps, difficult dentition).
 - (c) Gastric nausea. (Helminthiasis?)
 - (d) Affections of the pancreas.
 - (e) Hysteria, exophthalmic goitre.
 - (f) Intoxications (vegetable poisons, alkalies, mercury).
 - (g) Certain central neuroses.
 - (h) Inframaxillary neuralgia.
2. *Slight Secretion of Saliva*:
 - (a) (Sometimes with acid reaction of the fluid of the buccal cavity). Physiologic in infants during the first few months of life.
 - (b) Paralysis of facial and trigeminus nerves (anæsthesia, dysphagia, deflection of the uvula).
 - [(c) Mumps.]

23. THE MOST IMPORTANT CHANGES IN THE TONGUE

(May participate in affections of the rest of the buccal mucous membrane, *q.v.*)

1. *Macroglossia* (lymphatico-cavernous, fibrous, and muscular) and *Prolapse of the Tongue*.—Congenital malformation, myxœdema, monogoloid, acromegaly; after inflammatory processes and traumata.

2. *Congenital Fissure of the Tongue.*

3. *Adhesion of the Tongue, Ankyloglossia* (congenital [rarely acquired; cicatricial]).

4. *Coated Tongue* (in the first few weeks of life a delicate whitish coat is physiological):

(a) Nearly all acute infections, especially typhoid (fuliginous, triangular, edge and tip free), scarlet fever, pneumonia, diphtheria and non-specific anginas, dysentery, influenza (grayish white, dense, porcelain-like).

(b) Nearly all affections of the buccal cavity (often filthy coat), many gastro-intestinal conditions, dyspepsia, gastritis, etc.

5. "*Geographical*" *Tongue* (of young children) (Marking rapidly changing; no ulceration).—Latent tuberculosis; lymphatic diathesis.

6. *Discoloration of the Tongue.*—Cyanosis (simulated through coloring articles of nutrition and diet).

7. *Smooth Atrophy of the Tongue.*—Syphilis (affects particularly the circumvallate papillæ and the entire posterior half of the surface).

8. *Segmentation of the Tongue.* Diabetes mellitus.

9. *Edema of Papillæ of Tongue.*—Stomatitis in typhoid and scarlet fever ("strawberry and raspberry" tongue recognizable after desquamation of the coat on the third or fourth day), etc. Diabetes mellitus. Lymphatic leukæmia. Status thymicus.

10. *Ulcers of the Tongue.*—Primary conditions of the rest of the buccal mucous membrane, like stomacaces (ulceration at the edges, and notch formation), decubital from carious teeth. [Syphilis (penetrates into deep layers without reaction), tuberculosis (at the tip, torpid fissure with walled-up rims, ganglia).]

11. *Papules of the Tongue.*—(Recurring) hereditary and acquired syphilis.

12. *Ulcer of the Frænum* (A small growth, or erosion, about the size of a bean, in transverse position, lancet-like in shape, often with pseudo-membranous deposit at the frænum; occurs almost without exception only when the lower medial incisors are present).—Whooping-cough, much rarer in simple bronchial catarrh and other cough conditions.

13. *Dryness of the Tongue.*—Mouth-breathing, septic conditions, purulent peritonitis.

14. *Paralysis of the Tongue* (Motor insufficiency, possibly with lateral deflection, atrophy,—rugged surface,—disturbance of mastication and articulation). See No. 160 and following.—Paresis and paralysis of the hypoglossal nerve (protruded tongue is strongly deflected toward the affected side). Paresis and paralysis of the facial nerve (protruded tongue is deflected toward the well side). [Rigidity of the tongue in tetanus.]

15. *Tremor of the Tongue*.—Typhoid and other severe infections. Disseminated sclerosis. Bulbar paralysis. Beginning of progressive paralysis.

24. CHANGES IN THE FLOOR OF THE BUCCAL CAVITY

(Edema under the Tongue)

1. *Ranula*.—(Indolent, growing slowly, fluctuating, without inflammatory reaction, mostly unilateral or asymmetrical).

[2. *Sublingual fibroma* (Warty granulating tumor of traumatic origin after ulcer of frænum; frequent in Italy).]

3. *Angina Ludovici*.—In diphtheria, scarlet fever, measles; but also as independent ulcerative inflammation of the cell tissue (without involvement of the salivary glands) (Painful mesially, or symmetrically situated swelling; œdema, dysphagia, fever).

4. *Inflammations of the submaxillary and sublingual salivary glands*.—Independent and with parotitis.

25. CHANGES IN THE TEETH AND GUMS

A. *TEETH*.—The milk-teeth in children who are fed artificially are generally, *ceteris paribus*, weaker than those of breast-fed children.

1. *Increased Vulnerability*.—Syphilis, rachitis, and other conditions interfering with the constitution. Heredity. Diabetes mellitus.

2. *Circular Caries of the Neck* (of the upper incisor and bicuspid milk-teeth after green coloration).—Especially scrofulosis.

3. *Erosions of Enamel* in the form of stripes and rings at edge and crown of remaining teeth. Especially rachitis.

4. *Abnormally Small*.—Rachitis, syphilis, myxœdema, mongoloid, and various dental diseases.

5. *Malposition*.—Hypo-staphyly, adenoid vegetations, hare-lip, macroglossia, rachitis, degeneration.

6. *Hutchinson's Deformity* (Almost always in second dentition; inner upper incisors small, with spaces between them; at their edges, especially anteriorly, gnawed or cut in semi-lunar form. Pathognomonic, if only these teeth are affected). Hereditary syphilis (especially lues tarda).

7. *Loosening and Shedding*.—Infantile scurvy, several kinds of stomatitis, especially the ulcerous, leukæmic, and toxic (mercury). Gangrenous inflammation of gums in septic newborn and nurslings; also almost all severe acute and chronic pathological conditions.

B. GUMS.

1. "*Gingivitis*":

(a) Gums spongy, bleed easily, swollen, detached from rims of teeth.

B. GUMS.—Continued

- (b) Acute infections running a severe course, such as typhoid, diphtheria, measles, scarlet fever.
 - (c) Difficult dentition.
 - (d) Ulcerative stomatitis.
 - (e) Infantile scurvy (hæmorrhagic granulations, fetid odor).
 - (f) Congenital heart-disease.
 - (g) Acute lymphatic leukæmia.
 - (h) Gangrenous "osteogingivitis neonat." (before teething).
2. Lead Border, Bismuth Border.

26. ABNORMAL DENTITION**RELATIVE TIME CONDITIONS OF PHYSIOLOGICAL TEETHING. ERUPTION OF MILK-TEETH ("FIRST DENTITION").**

First period.....	$\left\{ \begin{array}{c c} & \\ \hline a & a \end{array} \right\}$	5-8 month; 2 teeth at end of period.
Second period.....	$\left\{ \begin{array}{c c} a' & a \\ \hline a & a' \end{array} \right\}$	8-12 month; 6 teeth at end of period.
Third period.....	$\left\{ \begin{array}{c c} c & a' & a \\ \hline c & a' & a \end{array} \right\}$	12-16 month; 12 teeth at end of period.
Fourth period.....	$\left\{ \begin{array}{c c} c & b & a' & a \\ \hline c & b & a' & a \end{array} \right\}$	18-24 month; 16 teeth at end of period.
Fifth period.....	$\left\{ \begin{array}{c c} c' & c & b & a' & a \\ \hline c' & c & b & a' & a \end{array} \right\}$	24-30 month; 20 teeth (complete milk teeth).

Thus, after the completed (6th to) 8th month one tooth erupts on the average every month.

Mnemonic formula for the approximate number (z) of teeth of an (m) months old baby in the milk-teeth period: $z=m-6$.

ERUPTION OF THE REMAINING TEETH ("SECOND DENTITION").

The first 4 molars.....	(d)	5th- 7th year.
The 4 inner incisors.....	(a)	8th year.
The 4 outer incisors.....	(a')	9th year.
The 4 anterior bicuspsids.....	(c)	10th year.
The 4 canines.....	(b)	11th-13th year.
The 4 posterior bicuspsids.....	(c')	12th-15th year.
The 4 second molars.....	(d')	13th-16th year.
The last 4 molars (wisdom teeth).....	(d'')	16th-26th year.

1. Delayed dentition. (Considerable delay and pathological interruption, *atypical course*, long persistence of an uneven number). (Moderate delay occurs in the healthy).—Rachitis, myxidiocy, mongoloid, cretinism, tuberculosis, syphilis, hereditary disposition.

2. Early dentition.—Hereditary disposition (of no importance), hydrocephalus?

3. Difficult dentition.

27. CHANGES IN THE MUCOUS MEMBRANE OF THE MOUTH

(Anterior Portions)

Demonstration: *For inspection see No. 31.*

A. HYPERÆMIA OF THE MUCOUS MEMBRANE OF THE MOUTH.

Coating and œdema of papillæ on the tongue.

1. *Red Spots*, symptom of "stomatitis erythematosa", "enan-thema":

(a) Later sometimes formation of a most delicate veil-like coat of desquamated epithelium ("stomatitis erythemopultacea"). Salivation; rarely considerable swelling of the glands.

(b) Many acute infections, partly prodromal:

(a) Scarlet fever (Dusky sprinkling, although predominating in the posterior portions).

(β) Influenza (Superficial, disseminated, dry reddening).

(γ) Measles (Jaggy, sometimes confluent red spots with central nodules, widely disseminated).

(δ) Mumps.

(ε) Sepsis neonatorum.

2. *Diffuse Œdema* (More or less disseminated, symptom catarrhal stomatitis. Later a whitish coat, slightly adherent and transparent. Salivation, slight swelling of the glands; physiological in children during the first week of their existence. Accompanying inflammatory (infectious) diseases, especially those of the fauces and the gastro-intestinal canal (typhoid, cholera infantum).

(a) Local irritation, trauma through bad teeth, difficult teething, scalding, corrosions.

(b) Proliferation of *Oidium albicans*.

[(c) Diabetes mellitus. Atropin intoxication.]

B. REDNESS, with whitish, slightly raised, minute sprinkled spots.

Simulated by particles of milk and epithelial desquamation.

1. *Incipient Thrush* (Beginning on the tongue; later especially on the cheeks, whitish points, difficult to wipe off, consisting of fungoid growths on the inflamed mucous membrane; are often grouped together, forming a dense creamy layer. Frequent in young nurslings having a digestive disturbance and with newborn without such disturbance; seldom with older children—tuberculosis, typhoid).2. *Koplik's Spots*: prodromal of measles (Somewhat easier to wipe off, mostly sparce, size up to a pin-head, consisting of epithelium and detritus, situated at the malar surface, behind the angles of the mouth and at the lips, surrounded by red areolæ).

C. (Multiple) SPECKLED, whitish yellow, delicate, flat, roundish, areolar deposits or discolorations with a tendency to further but only superficial expansion. No fetid odor. Glandular swelling and salivation moderate.

Might possibly be simulated by the whitish discoloration at some places of the buccal mucous membrane of newborn (alveolar border, hard palate), which is caused by the tension anæmia when opening the mouth.

1. *With a Distinct Preliminary Vesicular Stage*, symptom of vesicular stomatitis:

- (a) Epidemic aphthous stomatitis, (Foot-and-Mouth Disease). (Round, opalescent vesicles or spots, plaques, not grouped, rather large; mostly serious general manifestations, hæmorrhages, glandular tumors with involvement of the skin; ingestion of raw milk of diseased animals, mingling with same.)
- (b) Herpetic stomatitis.—Herpes-enuanthen (Fever; numerous vesicles or specks, always grouped, not larger than a pin-head; mostly along with similar ones on the tonsillar mucous membrane, and characteristic efflorescences on the lips.
- (c) Enanthemic varicella (usually only a few whitish plaques on lips and palate—also tongue—skin!).
- [(d) Impetigo and benign pemphigus neonatorum. (Always situated quite anteriorly, not grouped, rather large, tendency to tumefaction; up to then afebrile, skin manifestations!).]

2. *Without any (demonstrable) Preliminary Vesicular Stage*, symptom of maculofibrinous stomatitis.

- (a) Simple maculofibrinous stomatitis, idiopathic (alias aphthous stomatitis).—Aggravated dentition as a predisposing factor.
- (b) Diffuse maculofibrinous stomatitis. Typhoid, measles, scarlet fever, cerebrospinal meningitis, leucæmia (gonorrhœic infection).—Hæmorrhagic diathesis.

D. Very COHERENT, dense, FIBRINOUS white COAT, firmly adherent at first, later desquamated: symptom of pseudomembranous stomatitis.

May possibly be simulated by corrosions or scalding.

- 1. *Diphtheria* (Predominant at the labial mucous membrane, which generally appears covered in its entire extent with a lardaceous coat; only with faucial diphtheria).
- 2. *Pseudo-Diphtheria of Epstein*, in septicæmic newborn and nurslings (Fibrinous exudation generally starting from

D. Very COHERENT, etc.—Continued

the soft palate, rapidly progressing everywhere, forming a continuous grayish yellow, dimly lustrous, well marked, membranous deposit. General condition serious, death before involvement of the larynx).

E. (Multiple) EROSION OF MUCOUS MEMBRANE (Epithelial defect with reddened edges and discolored base).

1. In newborn at palatal raphe and immediate neighborhood: erosions after Bohn's milia (Without serious local or general disturbance, generally minute, lancet-like, sometimes larger in the form of a butterfly, symmetrically arranged at both sides of the median line).
2. In newborn and nurslings at the palatal corners: Bednar's aphthæ=Ulcers pterygoidea (mostly symmetric over the pterygoid ramus).

F. TRUE ULCER FORMATION on the mucous membrane, generally emanating from the dental gums, with fetid odor and considerable glandular swelling, often with notched tongue: symptom of "stomatitis ulcerosa."

1. *Idiopathic. Stomacacea* (Almost always commencing at the inferior maxilla; occurring only in children with teeth; blood-stained salivation; fusiform bacilli and spirochæte according to Plaut, Bernheim, and Vincent).
2. *Associated with Acute Infections*: diphtheria, scarlet fever, dysentery, typhoid (follicular ulcers at palate), malaria; also following after measles.
- [3. Mercurial stomatitis, infantile scurvy, and scurvy (blue-red ridge of the gums, bloody suffusion, early loosening of teeth; in scorbutus deep ulceration, in infantile scurvy (benign) phosphorus necrosis, botulism.)
- [4. Tuberculosis, syphilis, leukæmia, diabetes mellitus.]

G. (Primary) GANGRENE OF TISSUE, starting from the mucous membrane (or the dental gums), with ulceration and hæmorrhagic infiltration: symptom of "gangrenous stomatitis."

1. *Noma* (Gangrene commencing at the inner malar surface, very rapid extension; always unilateral; soon serious general condition, cadaveric odor).—Especially after measles, typhoid and other cachectic diseases.
2. "*Osteogingivitis Gangrænosa Neonatorum*" Klementowsky, a "gangrenous inflammation of gums during the first nursing period" (In children before the eruption of teeth, with shedding of tooth-crowns from the superior maxilla, generally without fetid odor, no salivation; gravest septic general condition).

H. PLAQUE FORMATION (Flat, bedlike-eminences of the size of a bean, with a grayish white macerated covering of the mucous membrane; stomatitis condylomatosa).—Syphilis (hereditary and acquired, recurring).

28. CHANGES IN THE PALATE

(exclusive of the involvement of the palatal mucous membrane in buccal and pharyngeal conditions)

1. *Torus palati*, exostosis medio-palatina (Protrusion of the pharyngeal raphe).—Idiocy, degeneration.
2. Hypo-staphyly (High, pointed bulging of the palate in the frontal and sagittal fissures).—Rachitis, adenoid vegetations, myxœdema, mongoloid, micromelia.
3. Palatoschisis (Lateral cleavage of the hard palate, median of the soft palate).
4. Perforation of the palate in syphilis, of the *soft palate* (if necessary with excision of the uvula) in diphtheria, and after measles, scarlet fever, etc.
5. Dotted hæmorrhages of the mucous membrane in meningitis, tuberculosis, cerebrospinal meningitis, hæmorrhagic diathesis, blood diseases, infantile scurvy, etc.

29. FETOR FROM THE MOUTH AND BREATH

Simulated by ozæna, gastric affections with foul-smelling eructations, helminthiasis.

1. Catarrhal stomatitis, aphthous stomatitis, dental caries, angina (faint, noticeable only in close proximity, disappears after washing the stomach).
2. Ulcerative stomatitis (characteristic, objectionable, fetid).
3. Noma, severe diphtheria (cadaveric).
4. Scarlet fever, diphtheria ("gluey," faint).
5. Pernicious anæmia (cadaveric).
6. Bronchiectasis, pulmonary gangrene (fetid).
7. Diabetes mellitus and coma, typhoid and scarlet fever, cyclical vomiting, autotoxic acute intestinal conditions (fruity, acetone odor).
8. Uræmia (like NH_3).
9. Intoxications with hydrocyanic acid, phosphorus, alcohol, chloroform, petroleum, acetic acid (odor of these poisons).
10. After vomiting (lactic and butyric acids).

30. INABILITY OR AWKWARDNESS IN SUCKING OR MASTICATING

1. General debility, premature birth, congenital and acquired debility.
2. Disturbances of innervation of the chewing and suckling musculature.—Central, basal, bulbar, peripheral paralyses of the trigeminus,

hypoglossal, and facial nerves. Bulbar paralysis, lesion of the cortical centre of association (frontal cerebrum), tetanus.

3. Infiltration, painful tension, and rigidity of the walls of the buccal cavity.—Sclerema, affections of the buccal mucous membrane and the sublingual region (*e.g.*, thrush, angina Ludovici), retropharyngeal abscess, mumps, otitis media, Thomsen's disease.

4. Factors forming a direct mechanical impediment.—Macroglossia, palatoschisis [harelip], ranula. Anomalies of the frænulum no impediment.

5. Use of the mouth in respiration.—All affections causing dyspnœa. Stenoses of the nose and pharynx (*see Nos.* 32, 65).

6. Psychic defects and cerebral conditions with disturbance of consciousness.—Idiocy (initial symptom!).

31. CHANGES IN THE MUCOUS MEMBRANE COVERING THE PALATAL TONSILS

(and in their neighborhood—palatal pillar, uvula, pharyngeal wall, pharyngeal tonsil)

Inspection of the throat should never be omitted, especially in feverish children; in obstreperous patients, however, it should be left until the end of the examination. Frequently fixation of the head is necessary. A child is placed on the lap of an adult, who presses the child's head against his shoulder. The best obtainable light is essential—the lightest window or artificial light—at the back of the physician, or held near his right ear. Generally a spatula or spoon-handle is required; the mouth gag may be dispensed with. The confidence of the patient should be obtained by careful insinuation; first examine the labial, malar, and anterior lingual mucous membranes, asking occasionally whether "it hurts." Gradually penetrate lower down, have "a" intonated, and finally depress the base of the tongue. Generally a retching effect is produced. When this is at its height, the lateral faucial walls bulge forward, causing the palatal tonsils to present their medial surface to the observer. The art of detecting and correctly interpreting in a few moments everything of importance is acquired by practice.

A. **HYPERÆMIA, EDEMA, SOFTENING** (Sometimes granular and œdematous: symptom of simple catarrhal sore throat. Only slight mucous filmy coat, if any. Slight glandular swelling).

1. *Uniform Diffuse Hyperæmia:*

(a) Independent catarrhal angina, acute and exacerbating in constitutional chronic catarrh (scrofula, lymphatic diathesis).

(b) Accompanying and symptomatic catarrhal angina in acute infections: Articular rheumatism (torticollis, dysphagia). Influenza (porcelain-coated tongue,

A. HYPERÆMIA, etc.—*Continued*

- coryza, and cough), erysipelas (purple color, skin manifestations), parotitis (protrusion and hyperæmia of mucous membrane at the excretory duct), urticaria (œdema, rapid appearance), typhoid, glandular fever, whooping-cough, appendicitis.
- (c) In intoxications: Solanaceæ, atropine (dry, dusky red), botulism, mercury, iodine, salicylic acid (catarrhal).
- (d) In affections of the tonsillar substance, especially erysipelatous and purulent tonsillitis (grave local manifestations, pain, dysphagia, œdema, formation of tumors and abscesses).

2. *Hyperæmia, Spotted or Punctated* (possibly confluent later).
—Pharyngeal Enanthem:

- (a) Acute infections; Measles (a few ragged spots, as an early symptom, 2 to 3 days before exanthema, together with Koplik's spots and striated hyperæmia or gauze-like malar coat; catarrhal syndrome), German measles (generally without pronounced catarrhal manifestations), scarlet fever (deep dark-red punctation of the soft palate on the first day of illness; sharply cut off at the hard palate, sometimes with hæmorrhages), cerebrospinal meningitis (hyperæmic spots on uvula, palatal pillars and tonsils), whooping-cough, influenza.

[(b) Tuberculosis (granulated surface).]

B. MUCOUS MEMBRANE as above, but covered with SPOTTED or PUNCTATE, generally well demarcated PATCHES OF WHITISH OR YELLOWISH COLOR, of pulpy consistency, which can be wiped off or crushed: symptom of "angina punctata". Patch has little or no tendency to expand; glandular swelling is entirely or almost absent.

1. *Simulated by Fungoid Growths on the Tonsillar Mucous Membrane* (thrush, leptothrix); e.g., cryptic pharyngomycosis (afebrile course, of long duration, no inflammatory reaction, accompanied by almost no complaints); and by chronic parenchymatous (submucous) conditions in the tonsil (well demarcated oval spots glimmering white or yellow in the middle of the tonsil).
2. *Follicular Tonsillitis* (rather uniform yellowish spots the size of a pin-head, scattered like stars in the sky, 6 to 10 in number; ulcerated follicles; in the early stage still firmly adhesive. Finding limited to tonsils; beginning of disease often brusque, with high fever and vomiting. Rather rare!).

B. MUCOUS MEMBRANE, etc.—Continued

3. *Angina Lacunaris*=cryptica (pultacea), acute, less frequently chronic (irregular shapes, whitish, from congested secretion in the lacunæ of the mucous membrane; always soft, pultaceous; coats grow from the depth. Frequent).
4. *Angina Punctata in Infections*: prodromal or long preceding in articular rheumatism, typhoid (follicular form); accompanying scarlet fever, parotitis.

C. MUCOUS MEMBRANE IN SHARPLY CIRCUMSCRIBED UNIFORM PATCHES, dimmed, whitish discoloration or (rarely) vesicular detachment: symptom of "*angina maculofibrinosa*." Simulated by scalding and corrosions.

1. *Aphthous angina*=*angina maculofibrinosa* (rare, with aphthous stomatitis, *q.v.*).
2. *Herpetic angina*=(after a very short vesicular stage, sharply demarcated, round, disk-like deposits, seldom confluent, pseudomembranous. Often begins with high fever, etc. Concomitant herpes labialis is no proof of the nature of the disease, as this also occurs in diphtheria).
3. *Enanthema Varicellarum* [et variolæ] (together with exanthema and enanthema at the buccal mucous membrane).

D. MUCOUS MEMBRANE as above (see A.), besides COVERED BY A COHERENT MEMBRANOUS LAYER, which remains firmly attached for a long time: symptom of "*angina pseudomembranacea*." Simulated by corrosion scabs after poisoning and painting; by thrush and leptothrix proliferations (characteristic bacteriological findings); by a traumatic layer after tonsillotomy (afebrile, previous history; no tendency to expansion).

1. *Pronounced Expansive Tendency of the Pseudomembranes*:

- (a) Diphtheria, pure or scarlatina-mixed infection (more than 90 per cent. of all real pseudomembranous layers are of a diphtheritic nature; grave general manifestations, generally rather high fever, albuminuria, considerable early glandular swelling; creeping over to palatal pillars, lateral pharyngeal wall, and uvula; involvement of nose and larynx; first occurs on convex surfaces of the tonsils; exterior circumstances: opportunity for infection, (infection from playmates).
- (b) Scarlet fever (without diphtheria), pseudomembranous form (coat as such scarcely distinguishable from diphtheria; distinction through concomitant circumstances and negative bacillary finding).

D. MUCOUS MEMBRANE, etc.—Continued

(c) "Diphtheroid" (cannot safely be distinguished, generally takes a benign course, unless it has appeared after measles and scarlet fever; tendency to recurrence; no characteristic bacterial findings).

2. Tendency of the Pseudomembranes to spread, Absent or Slight:

(a) Diphtheria-like angina in infections: Preceding typhoid (prodromal) and in the course of typhoid. Accompanying ulcerous and phlegmonous angina. Septicæmic pseudo-diphtheria of Epstein (in newborn).

E. MUCOUS MEMBRANE eroded or ULCERATED, suppurative, discolored coat: symptom of "angina ulcerosa."

1. *Ulcerative Tonsillitis* (tonsillitis ulceromembranosa) Plaut-Bernheim-Vincent (severe—often semilateral—local, few general symptoms; with ulcerous stomatitis; characteristic fetor and bacterioscopic findings; glandular tumors; ulcers round, with gray coat, and walled-up brim, often rather indurated, chancre-like).

2. *Severe Scarlatina Angina*, scarlet-diphtheroid (generally appearing from the third to the fifth day of illness—rarely later, as secondary scarlet-diphtheroid—with dense discolored layers, under which deep ulcers attack tonsils, pillars, and uvula. Nasal flow; often laryngeal stenosis through œdema, large and painful glandular tumors).

3. *Diphtheritic Tonsillitis*, malignant form, especially after measles and other infections (after desquamation of the membranes, suppurating ulcers remain behind).

F. GANGRENOUS DISINTEGRATION OF MUCOUS MEMBRANE (Tonsil covered by necrotic, discolored (generally grayish green) shreds of tissue and deposits; fetor: symptom of "angina gangrænosa").

1. *Severe Diphtheria*.

2. *Angina Scarlatina Necroticans*.

3. *Termination of Non-Specific Anginas* in other diseases weakening the constitution (typhoid, measles, etc.).

NOTES CONCERNING DIPHTHERIA AND SYPHILIS

1. *Diphtheria* can always present all its various types (the catarrhal (alleged to be rare), the punctated, and the ulcerous forms), and this is to be kept in mind even when there are no pseudomembranes and none forming. The assumption of having to deal with diphtheria is supported by the involvement of pharynx, nose, and larynx, with considerable glandular swelling, albuminuria, severe general conditions, and oppor-

tunity for infection; also by the result of examination of the membrane: firm, fibrinous condition, not destroyed by rubbing, few bacteria, demonstration of the Löffler bacillus.

Diphtheria is not excluded by absence of fever, negative or dubious result of bacteriological examination, demonstration of other germs, alleged second attack and absence of discomfort.

Bacteriological demonstration of diphtheria (staining and streak culture): Portions of the deposit are taken from the fauces by means of a spoon forceps, washed with water, transferred to coagulated cattle serum (after Löffler, for purposes of culture), and crushed between slides (for fixation and staining with Löffler blue). Attention should be paid to the coherence of the mass, to the rapid and luxuriant budding, color, shape of colonies, stratification, granulation, and staining of the bacilli. In the absence of faucial deposit use nasal secretion, sputum, mucus from the laryngeal tube, or coughed up membrane.

2. *Syphilis* exhibits on the tonsillar membrane a unilateral primary affection (rare) of papules and gummata. The papules often differ only slightly from the maculofibrinous type of angina and may also simulate pseudomembranes. The gummata are ulcerative. Diagnosis through torpid course, glandular tumors, and accompanying circumstances.

32. PHARYNGEAL STENOSIS

Mouth-breathing, snoring, and gurgling respiratory murmur, dyspnoea of a pharyngeal character, inspiratory stridor and often a fluctuating respiratory murmur, dulness of hearing, anginous voice, backward and lateral position of the head, dysphagia. Paroxysms of pseudocroup, spasms of the glottis and asthma as reflex manifestations. Disturbance of articulation of "m" and "n". Habitual mouth-breathing leads to deformities of the cranial skeleton; hypo-staphyly, card-position of the teeth, depression of the bridge of the nose; also to exophthalmos and adenoid expression, aprosexia, etc.

Demonstration: Digital examination of the rhinopharynx. The physician, standing on the right side behind the sitting or standing child, fixes the head with the left hand by pressing it against his body, introduces the cleansed and short-nailed index finger of his right hand into the patient's mouth, and proceeds further up to the soft palate, his finger being first stretched and then curved hooklike. A mouth gag is never required in toothless children, and seldom in others, provided the procedure is executed quickly and skilfully, as the retching reflex prevents the possibility of biting.

Simulation of tumors or swellings in the rhinopharynx is possible if the uvula was turned over, causing the protrusion of the lateral pharyngeal walls by way of reflex spasm, and also by the ridge of the atlas.

Pharyngeal stenosis is simulated by stenosis of the nose and the lower air-passages, especially by compression of bronchial glandular tumors. *For differentiation, see Dyspnœa.*

A. ACCIDENTAL (OR ACCIDENTALLY EXACERBATED, ACUTE) PHARYNGEAL STENOSIS.

1. *Acute Inflammatory Affections of the Palatal Tonsils and Pharyngeal Walls:*

Angina phlegmonosa and gangrenosa "idiopathic" and metastatic. (Typhoid, septic pyæmia.) Tonsillar, retro- and paratonsillar abscesses.

More frequent in older children, change of shape and position of tonsil, displacement of uvula, tumor shortly fluctuates, semilateral swelling of the neck between inferior maxilla and sternomastoid. Fever, severe subjective complaints; pain; difficulty in swallowing.

Retropharyngeal abscesses, mostly acute, ulcerative lymphadenitis; buccal infection, after angina, otitis, or metastatically after acute general infections (scarlet fever, sepsis, influenza, pyæmia), spondylitis.

Chiefly in children between 6 and 24 months of age; following coryza, angina, or pharyngitis. Fever. Tumor, soon fluctuating, often visible, always palpable, always situated laterally. Continued dyspnœa, occasionally exacerbated, depending on position of body; often rough cough, dysphagia, stiffness of the head.

Acute pharyngeal angina (sometimes of diphtherial nature) recurring (in adenoid vegetations).

Without posterior rhinoscopy, few objective signs, except those of the acute febrile stenosis generally and glandular swelling at the neck. Pains radiating toward the ear; secretion from fauces and nose, ulcerotenacious deposit on posterior faucial wall.

B. HABITUAL (CHRONIC) PHARYNGEAL STENOSIS.

1. *Chronic Hyperplasia of the Palatal Tonsils.*—Scrofula, lymphatic diathesis, status thymicus, mongoloid, recurring anginas, tonsillar tuberculosis, dental caries, pseudo-leukæmia and lymphatic leukæmia. Also hereditary and congenital.
2. *Chronic Hyperplasia of the Pharyngeal Tonsils, Adenoid Vegetations* (Aprosexia, deformities of the facial skeleton through habitual mouth-breathing, also of the thorax; digital examination).—In the same conditions as the above.
3. *Chronic Retropharyngeal Lymphadenitis and Cold Abscess* (Position of head. Doughy swelling at neck and nape, generally no fever, few subjective complaints).—Tuberculosis of the glands, vertebral caries.
- [4. *Macroglossia, ranula, polypi of the rhinopharyngeal space* (only in older children, a hard, smooth, slightly movable tumor, palpable; epistaxis).—True tumors of the tongue (lipoma), of the fauces (polypus), of the tonsils, of the alveolar process, and of the base of the cranium (sarcoma).]

33. DYSPHAGIA

Interference with the act of swallowing: inability to swallow, swallowing the wrong way, regurgitation and retching in swallowing, pains in swallowing.

Simulated by coryza and other nasal stenoses in nurslings, anorexia, dread of drinking in pyloric stenosis, mouth affections, parotitis.

Congenital and acquired debility, also:

A. AFFECTIONS OF THE PHARYNX.

1. *Disturbances of Innervation in the Pharyngeal Region* (tongue, soft palate, pharynx; trigeminus, glossopharyngeus, vago-accessorius, hypoglossus and facial nerves).

(a) Cerebral conditions, especially of pons and medulla (hæmorrhages, embolism, tumors, encephalitis, sclerosis, kernaplasia, ascending spinal conditions). (See also *Bulbar Palsy*, No. 158.)

(b) Basal conditions (meninges, tumors, traumas).

(c) Peripheral lesions (neuritis, post-diphtheritic, toxic, etc. Injury through bronchial glandular tumors, exudative pericarditis, etc.).

Paresis or Paralysis of

Trigeminal nerve.
Facial nerve.
Vago-accessory nerve, (pharyngeal plexus).
Glossopharyngeal nerve.

Regurgitation through the nose (also nasal voice and nonraising of the soft palate in intonation). Sticking fast in the pharynx. Gliding into the larynx (swallowing the wrong way).

[(d) Thomsen's disease.]

- (e) Increased irritability (spasms of deglutition). Trismus and tetanus neonatorum; hysteria (globus), chorea minor [Lyssa, intoxication by strychnia and solanaceæ.]

2. *Factors Constituting Direct Mechanical Impediment:*

- (a) Pharyngeal conditions accompanied by considerable swelling or rigidity of the soft parts. Inflammatory conditions of all kinds, retropharyngeal abscesses, anginas; also macroglossia (myxœdema), œdema in serum-disease, after corrosion, sclerema, and sclerœdema neonatorum.
- (b) Factors causing stenosis. Retropharyngeal abscess. Thyroiditis and strumitis, pharyngeal (and laryngeal) tumors and foreign bodies, laryngeal perichondritis, pseudo-leukæmic and other glandular tumors, hypertrophy of the palatal tonsils, adenoid vegetations, extreme rigidity of the neck.

B. AFFECTIONS OF THE ŒSOPHAGUS.

1. *Acute Conditions:*

- (a) Corrosion through heat, brine, acids, foreign bodies (often without alarming manifestations and pain), inflammation in thrush (also occlusion by fungoid masses), scarlet fever, diphtheria, measles, ulcerous stomatitis, mercury poisoning.

34. VOM

Simulated by regurgitation.

		<i>Cerebral vomiting</i> without apparent cause, without nausea, retching, or effort. Large quantities in gushes; gives no relief; therapeutic measures fruitless; occurs frequently in certain positions of the body. Masses mucowatery; abundant HCl; presence of cerebral symptoms, generally absence of gastric symptoms, frequently constipation.¹		<i>Gastro-enteric and peritoneal vomiting</i>, often following pallor and nausea, with retching and effort, which generally brings relief. Masses with undigested food remnants. Hypochlorhydria; mucus vomiting. Absence of cerebral, presence of gastric signs. Epigastric and abdominal pain.		<i>Prodromal and initial vomiting in (acute) infections.</i>	
Accidental, occasional.	In nurslings.	Hydrocephaloid. Serous meningitis and acute hydrocephalus.	In the course of otitis. Sinus thrombosis.	Invagination (often bloody and fecal masses). [Special condition of (breast) food.]	Acute and chronic gastro-enteritis. Dysentery. Typhoid. Indigestion.	Nearly all acute infections.	Erysipelas. Typhoid (not frequent, but oftener than in adults).
	In older children.	Brain tumor. Meningitis (lasting generally only a few days) cerebrospinal, suppurative. Encephalitis. Cerebral abscess. Concussion of the brain.	Hyperemia and hemorrhage of the brain, spinal cord, and meninges. Encephalitis. Cerebral abscess. Concussion of the brain.		Dyspepsia. All forms of intestinal obstruction, especially appendicitis, constipation, incarcerated hernia (stercoraceous). Foreign bodies in stomach, stone colics, acute and chronic peritonitis, (often biliary masses, also fecal).	Scarlet fever (almost constant with sudden onset). Croupous pneumonia (generally only on first day 1-3 times). Influenza, measles, diphtheria, parotitis, catarrhal icterus, malaria, purpura fulminans.	Anterior poliomyelitis. Cerebrospinal meningitis. Acute pneumonia. [Variola.]
Habitual, always recurring.	In nurslings.		Chronic hydrocephalus. Cerebral sclerosis.	Pyloric stenosis, spastic (without retching, straining or nausea, explosive, massy). Organic (as above, perhaps still regular. Masses often malodorous, free from bile. Occur in the first few weeks of life, uncontrollable, especially in breast-fed children; no HCl). Other congenital malformations, with stenosis of the digestive tract (generally grave general picture, deformities).	Atony and dilatation of the stomach (large masses, malodorous, long after meals). Habitual over-feeding (often without nausea or straining, "running over," milk still uncoagulated).		
	In older children.			Worms (especially when stomach is almost empty; vomited matters sometimes contains the parasites or their eggs).		Vomiting after meals in pulmonary tuberculosis (incipient).	

¹These symptoms of "cerebral" vomiting are not absolutely invariable; they may be absent in "cerebral" vomiting and be present in other forms.

VOMITING

See Dysphagia, No. 33.

<i>Vomiting in intoxications (of endogenous and ectogenous nature). Often masses of characteristic appearance and odor.</i>	<i>Vomiting after excitement; cough and violent sensory irritations.</i>	<i>Vomiting in cardiac insufficiency (vagus symptom); sometimes cerebral in character.</i>	<i>Vomiting in cases of general neuropathy.</i>	<i>Vomiting from other causes.</i>
Various vegetable poisons, metal salts, acids, alkalies, poisonous gases; many medicaments, especially all emetics and the expectorants in large doses, narcotics. Tainted articles of diet and nutrition. Uremia. Diabetes mellitus. [Cystopyelitis and incipient nephritis, even without albuminuria and other signs of uremia.]	Whooping-cough. Cessation of bronchial glands. Perforated empyema. Abscess of the lungs. Violent pains in anal fissure and constipation colic; stone in ureters and bladder; traumas, burns.	Myodegeneration and myocarditis in and after acute infections (diphtheria, typhoid). Exudative pericarditis.	Highly neurotic children vomit from the most trivial causes (psychic shock, ingestion of various articles of diet and nutrition or medicaments).	Acute pharyngitis. Irritation of the pharyngeal mucous membrane in examinations and operations. Acute hemorrhagic diatheses (Werthof's disease). Heat stroke.
Cyclic vomiting (violently and frequently, lasting for days, recurring periodically; acetone odor; no dyspeptic signs; ceasing spontaneously; in children over 3 years).	Chronic bronchiectasis.	Congestion of the large circulation in insufficiency of cardiac valves.	"Schoolsickness" (vomitus matutinus scholarium). "Neuro-arthritis." Hysteria (hyperemesis). Epileptiform attacks. Migraine (recurring in cycles). Gastric neuroses.	Chronic nasal and faucial affections; adenoid vegetations and hypertrophy of the palatal tonsils. All forms of chronic anemias; chlorosis, pernicious anemia, chronic leukemia. Stricture of oesophagus (no true vomiting).

The centripetal paths of the vomit reflex run in the vagus nerve, glossopharyngeus, and splanchnicus. The centrifugal paths of the vomit reflex run in the vagus nerve, phrenicus, splanchnicus, and in dorsal nerves. Vomiting centre in the medulla oblongata.

B. AFFECTIONS OF THE ŒSOPHAGUS.—Continued

- (b) Pericæso-phagitis (after Pott's disease, caseation of the bronchial glands, pleuritis, pericarditis, decubital ulcers of the larynx and in the trachea after tracheotomy and intubation), spasms in lyssa, tetanus, tetany, hysteria (in irritable children under different circumstances, occurring for instance in swallowing a pill), idiopathic spasm of the heart in neurasthenia.

2. Chronic and Habitual Conditions:

- [(a) Congenital atresia and stenosis of the œsophagus.]
 (b) Cicatricial stricture after corrosion, scalding, syphilis, trauma from foreign bodies (examination with a sound).
 (c) Ulcers as a result of compression (mediastinal glands, large thymus).
 (d) Diverticula (septic contents, regurgitation of such contents, result of sound examination).

34. SEE TABLE PRECEDING**35. HÆMATEMESIS (BLOOD VOMITING)**

Simulated by vomiting of blood from fissures of the maternal nipple (in breast-fed children), by epistaxis, hæmorrhages from mouth and fauces in violent cough and vomiting; by vomiting of red-colored articles of diet, hæmoptysis.

1. Hæmorrhages Originating from the Stomach:

- (a) Hæmorrhagic diathesis (hæmophilia, Werlhof's disease (purpura hæmorrhagica), Henoch's purpura, primary blood diseases, "hæmorrhagic" syphilis, sepsis), hepatic cirrhosis, congenital obliteration of the bile ducts, corrosive gastritis (especially by acids and brine), foreign bodies in stomach, gastric ulcers (round ulcer, tuberculosis, poisons).

2. Hæmorrhages Originating from the Intestines from analogous causes and especially in congenital and acquired intestinal occlusion. [Worms.]

3. Hysteria, Cyclic Vomiting, etc.**36. BILE AND STERCORACEOUS VOMITING**

All vomiting (with the exception of the pylorostenotic) may occasionally lead to the expectoration of biliary masses, especially in cerebral and peritoneal affections, also in pneumonia and beginning pleurisy. Stercoraceous vomiting in diffuse suppurative peritonitis and intestinal occlusion (*see No. 45*).

37. SINGULTUS, ERUCTATIONS

In healthy infants, especially breast-fed children, after nursing.

1. Affections of the central nervous system (medulla, pons).
2. Neuroses (hysteria, chorea minor).
3. Psychic excitation.
4. Irritation of sensitive nerves in various organs (stomach, intestine, renal pelvis).
5. Irritation of phrenic nerve.—Pleuritis, pericarditis, mediastinal tumor, peritonitis; affections of liver and pancreas.
6. Acute infections.—Cerebrospinal meningitis, whooping-cough.

38. STATUS GASTRICUS (ACUTE)

Syndrome of gastricism and indigestion; in children over 1 year.

Pallor of the face, headache, gastric pain, nausea, furred tongue, evening fever, change of stools.

1. True indigestion.—Especially the ingestion of excessive quantities of food difficult of digestion, and of tainted articles of diet (in most cases rapidly relieved by laxatives, emetics, and dieting).

2. Other gastric conditions: Catarrh of the stomach, pseudomembranous conditions in diphtheria, scarlet fever, corrosive gastritis, phlegmonous and thrush gastritis. [Round ulcer, tuberculosis, hæmorrhages.]

3. Intestinal conditions, especially appendicitis, invagination, parasites.

4. Intoxications (also gas poisoning).

5. Beginning of acute infections, especially pneumonia, influenza, typhoid, sepsis, polyarthritis, erysipelas, non-specific anginas and other affections, such as tuberculous meningitis, pleurisy with effusion, otitis media.

39. SYNDROME OF CHRONIC DYSPEPSIA

(frequently associated with the symptoms of atony of the stomach—in older children)

Anorexia, nausea, eructations, periodically recurring vomiting, thirst, coated tongue, feeling of pressure after eating, burning sensation in the gastric region, distention of the abdomen, irregularity of stool or constipation, flatulence, offensive breath, slight fever attacks, polyuria and perspiration, headaches, ill temper, fatigue, sallow complexion.

(Atony of the stomach: splashing sound, clapotage,—very extensive and pronounced after meals,—epigastric distention in cushion form, isochymia, eructations, heaviness and fulness in the abdomen, severe vomiting a long time after meals.)

1. Gastric and intestinal conditions, especially with irrational dietary regime, lymphatic diathesis, rachitis.—Gastro-intestinal catarrh, chronic appendicitis, invagination, intestinal parasites (diet test, fecal examination, palpation).

40. CHANGES IN THE STOOLS

	Normal (after the first few days of life).		Simple dyspepsia relative in (maternal) over-feeding, absolute in functional derangement (artificially fed).
	Breast-fed.	Artificially-fed. ²	
Number of stools per day....	1-2 (-3).		Varying (usually increased).
Total daily quantity (in comparison with ingestion of nutrition).			Increased.
Relative proportion of the single constituents. ³	Principal constituent: Secretions, up to 1/2 of the mass; bacteria, little feces.		Large quantities of feces.
Consistency ⁴	Soft.	A little firmer; paste-like.	Loose crumbs (increased albuminous remnants).
Appearance and most apparent ingredients.	Mostly homogeneous.	Mostly homogeneous.	Soapy glistening, softer, unctuous (mixed fat remnants), pastelike, frothy (starch remnants, carbohydrate fermentation in starch-feeding).
	Without apparent elementary constituents (macroscopic).		
Color (fresh stool)	Unicolored, yolk or gold yellow.	Unicolored, approaching sulphur yellow.	Unicolored, brown-yellow to brown (remnants of flour and malt), foamy, white-yellow (fat remnants).
Odor.....	Pleasant weak acid-aromatic.		Caseous, easily putrefying (albuminous remnants), scratching, rancid (fat remnants), zwieback-malty (flour remnants), after lactic acid, acetic acid (fermenting sugar).
Reaction (litmus).....	Weakly acid.	Mostly weakly alkaline.	Usually acid.
Microscopic analysis	Outside the bacteria, few or no distinct elementary constituents (detritus, epithelia, smallest fat acini, stearic acid needles, fat soaps, inorganic salts in crystals, usually no mucus), typical fecal flora.		Fat in puddles and masses of stearic acid needles; grouped remnants of cereals in large quantities; hairs, husks, starch corns. Change of quality of
Chemical analysis	Soluble protein substances, nucleo-albumin, and their metabolic products, fats, lactic acid (in breast-fed children).		Albuminous remnants or fat increased; starch; lactic acid; acetic acid.
	Bilirubin. ⁵	Bilirubin and urobilin.	Increased urobilin.
Biological analysis	Always human albumin		
Defecation.....	Apparently with comfort.		Discomfort or

¹It should be borne in mind that all the most important types of pathological conditions frequently run into each other and that often different pathological conditions obtain in various parts of the intestine.

²Diluted sugared cow's milk.

³Material ingredients of nurslings' stools:

1. Feces—undigested or half-digested portions of food;

SYMPTOMATOLOGY OF CHILDREN'S DISEASES 63

OF THE YOUNG INFANT¹

Conditions with catarrhal affections in the foreground,		Inflammatory processes, enterocolitis,	
in the higher parts of the intestine.	in the lower parts of the intestine (colon).	in the higher parts of the intestine.	in the lower parts of the intestine (colon).
Slightly increased.	Increased.	Increased.	Up to 20 and over.
Increased.	Increased.	Increased.	Increased.
Increased mucus masses and bacteria.		Principal constituent: inflammatory products and frequently greatly increased bacteria (up to two-thirds of the total quantity).	
Soft to fluid, incoherent, coarse, or thin-flaked dissociated; with roundish, whitish so-called casein flakes.		Fluid-watery, splashing.	Purulent-bloody; then again fecal; the different defecations vary greatly.
Intimately mixed with the feces is mucus, filamentous consistency.	Mucus badly mixed, like fruit jelly or frog spawn	3 layers. Fat pools and gray flakes of mucous membrane.	
Multicolored, varying, green-white, yellowish green.		Dirty gray to leek green.	Multicolored, red spots, yellow spots.
Sweetish.		Sweetish to malodorous.	Often ichorous, ammoniacal.
Varying; usually weakly alkaline.		Alkaline.	Alkaline.
"Formed mucus," transparent striated mucous paths in cylindrical form with cellular infiltrations; in the so-called casein flakes stearic and fat soap needles.		Sometimes bloody remnants, much intestinal epithelium.	Blood, pus, mucus carrying many cells.
Mixed fairly well.	Badly mixed.		
the picture of bacteriological vegetation.		Occurrence of pathogenic schizomycetes.	
Much mucus and food remnants.		Sometimes sugar directly demonstrable.	
Bilirubin (and urobilin).		(Bilirubin and) urobilin.	
only, animal albumin never demonstrable.			
pain preceding, under cooing, borborygmus, and unrest.			With pain and tenesmus.

2. Secretions—digested secretions or their constituents and desquamated epithelium.

3. Bacteria (living and dead).

⁴Stool collected on the diaper (consistency increased through absorption of water).

⁵The genuine bile pigment (bilirubin) is changed into urobilin upon occurrence of slight rotting processes in the intestine of older breast-fed and all artificially fed children, unless the evacuation of the intestine is greatly accelerated.

2. Anæmic conditions: in early infancy, especially after premature delivery; later in blood diseases; "school and growth diseases" (shortly before and during puberty, palpitation, anæmic condition of blood, dull pains in the long bones, vomiting in the early morning, often mental lassitude).

3. Latent tuberculosis.—Lengthened prodrome of tuberculous meningitis, chronic tuberculous peritonitis, pulmonary tuberculosis (temperature curve, general condition, opportunity for infection, diazo reaction, indicanuria).

4. Neuropathy:

(a) Chorea minor, gastric neurasthenia.—In floating kidney (neurotic and psychic signs).

5. Chronic interstitial nephritis and Pavy's disease (make examination of day urine).

40. SEE TABLE PRECEDING

41. DIARRHŒA IN OLDER CHILDREN *

Examination of the feces for the purpose of determining the presence of special ingredients is made by dissolving them in a large glass bowl of water standing on light and dark ground. Take samples for microscopic examination. Physiological condition: Formed, rather homogeneous, brown mass of pastelike consistency, containing only a few macroscopically distinguishable undigested particles of food, and little mucus; of characteristically fecal but not actually fetid odor.

A. INTESTINAL CONDITIONS (especially).

1. *Acute and Chronic Gastro-enteritis:*

(a) Of infectious nature.—Cholera nostras of second childhood; after stomatitis, angina, in malaria.

(b) Of toxic nature.—Metals, metalloids, uræmic enteritis, tainted articles of diet and nutrition; after taking cold.

2. *Appendicitis* (toxæmic, with septic peritonitis).

3. *Dysentery* (Very numerous stools, evacuated under tenesmus, containing distinct bloody mucus, and pus, and pathogenic germs; colon painful on pressure; often collapse).

4. *Tuberculosis of the Intestine (and Stomach)* (Very numerous, watery, offensive, often grayish black stools, containing mucus, pus, and blood, together with gray, granular bits. Make bacteriological examination. Swelling of inguinal glands; diarrhœa intermittent; pains moderate).

* In younger children diarrhœas as manifestations of other acute and chronic pathological conditions are too frequent to be semiotically of value.

A. INTESTINAL CONDITIONS (especially).—Continued

5. *Typhoid Fever* (In either the early or the later stages (beginning of second week) stools of pea-soup or coffee-and-milk color, disappearing in defervescence. Sometimes dysenteric).
6. *Amyloidosis of the Intestine*.—(Tuberculosis, caries, scrofula, hereditary syphilis).
7. *Intestinal Parasites (and Trichinosis)* (Mucoid and bloody evacuations frequent in infestation by trichocephalus and anchylostomum).
8. *Intestinal Lithiasis* (Mucus, blood, and sand in fluid stools; paroxysms of pain in colon; vomiting and fever).

B. GRAVE ACUTE INFECTIONS (Diarrhœa from the beginning in scarlet fever, etc., and in the later stages if the disease follows an irregular course and cardiac insufficiency sets in (vagus?); e.g., in measles, scarlet fever, diphtheria, influenza, sepsis, pneumonia, milary tuberculosis, erysipelas, cerebrospinal meningitis, malaria, anterior poliomyelitis, putrid infections, and non-specific anginas).

C. CERTAIN CONSTITUTIONAL DISEASES AND NEUROSES.—Severe anæmia, leukæmia, rachitis (stool frothy, mucous, massy, very malodorous). Exophthalmic goitre, hysteria.

D. PSYCHIC INSULTS (fright, terror).

42. CONSTIPATION

Simulated by absence of stools in pyloric stenosis, in abstention from food in consequence of anorexia, in difficulties of mastication, suckling, and swallowing (*q.v.*), and insufficient milk in the nurse.

A. ACCIDENTAL.

1. Loss of water of the organism in many febrile diseases; formation of cutaneous œdema (nephritis).
2. Partaking of constipating articles of diet, nutrition, and medication; absence of biliary function (catarrhal icterus).
3. Paralysis of the peristalsis (and abdominal pressure), through peritonitic and grave enteric conditions and through affections of the central nervous system. (Cerebral tumor, affections of the spinal cord, meningitis).
4. Psoas insufficiency and general bodily debility following infectious diseases.—Convalescence from influenza or typhoid.
5. Reflex and voluntary retention of the stool in (painful) anal diseases.—Fissure, ulcers, and eczema; spasm of sphincter.
6. Accidental intestinal obstruction (*q.v.*).

B. HABITUAL.

1. Congenital causes of mechanical impediment.—Stenosis (and atresia) of rectum, anus, and duodenum, adhesions, inflexions, congenitally large colon and Hirschsprung's congenital dilatation and hypertrophy of the colon (Obstinate constipation,—sometimes from birth,—considerable meteorism, balloon-belly; visible peristalsis of the intestines; abnormal capacity of the large intestine (demonstrated by palpation and injection of water); defecation seldom spontaneous, unless caused by secondary conditions of the mucous membrane; diarrhœas).
2. Gastro-intestinal atony.—Rachitis, myxœdema, cretinism, anæmia.
3. Gastro-intestinal affections; chronic dyspepsia, and intestinal tuberculosis; marasmus.—Exclusive milk, meat, or egg diet, dry diet, or food containing insufficient fat. Errors of regime in breast-fed children and in their nurses (*e.g.*, overeating); highly sterilized milk and prolonged exclusive milk diet in bottle-fed children.
4. Central and peripheral nervous affections, paralysis of intestine and abdominal pressure; chronic hydrocephalus, other chronic cerebral and spinal affections; neuritic paralysis.
5. Abulia (generally in defective education or in masturbation); other psychogenic pathological conditions.
6. Unsuitable mode of living—excessive sedentary habits (in school children), ill-regulated life.

43. SPECIAL CHANGES OF THE STOOLS IN OLDER CHILDREN

1. Feces grayish white, asbestos colored, clay colored, lustrous, soapy, stinking.—Acholia in catarrhal icterus, certain cirrheses, amyloid liver, chronic peritonitis, pancreatic affections, cholera, rachitis.

2. Feces light yellow, fluid, of penetrating odor, with fat drops, fat pools, and whitish soap particles, and with a greasy lustre.—Fat diarrhœa, *tabes mesenterica*.

3. Feces grayish yellow to greenish yellow, fluid, double stratification, frothy, stinking. Typhoid II.

4. Pus in feces.—Dysentery, intestinal ulcers, perforation of appendicular abscesses, enteritis.

5. Blood in feces, *see* No. 44.

6. Membranous masses of mucus in feces (simulated by food remnants, foreign bodies, and parasites.—Mucomembranous enterocolitis (in neuroses), diphtheritic and dysenteric colitis, invagination.

7. Tissue shreds in feces.—Ulcerous and gangrenous intestinal conditions, intussusception.

8. Sand in feces (simulated by foreign bodies).—Lientery, dyspepsia, and catarrhal conditions.

9. Charcot-Leyden's crystals in feces.—Especially in intestinal parasites.

44. BLOODY STOOLS

Blood, more or less changed and more or less mixed with fecal masses, depending upon its origin and the duration of its intestinal passage. Stool red, brown-red to tar color, often slimy, offensive. Proof by microscopical or chemical examination (hæmin test, aloin test). The latter test also demonstrates blood from ingested underdone meat, and under suitable circumstances will therefore also be found positive in the healthy.

A. IN NEWBORN CHILDREN.—*Melæna neonatorum* (simulated by blood swallowed from eroded warts or in epistaxis, by maternal blood taken in at birth, and by excretion of meconium,—the latter being demonstrated by the Gmelin test, and not by the chemical blood tests).

1. *Melæna Ulcerosa* (Collapse occurs mostly between the first and third day of life).
2. *Certain Forms of Sepsis* (Buhl's disease and others).
3. *Congenital Syphilis* (especially with hepatic affections).
4. *Hæmophilia*.
5. *Congenital malformations*. Congenital heart diseases, obliteration of the bile ducts.

B. IN OLDER CHILDREN.—Simulated through swallowing certain medicaments and articles of nutrition (*e.g.*, iron, bismuth, blackberries); epistaxis, beginning of menstruation.

1. *Hæmorrhagic Diathesis and Primary Blood Diseases*.—*Hæmophilia*, Henoch's purpura, purpura hæmorrhagica, infantile scurvy; amyloid degeneration of walls of vessels after chronic ulcerations; certain liver cholæmias and pancreatic affections(?); leukæmia, pernicious anæmia.
2. *Acute Infectious Diseases*.—Severe diphtheria, malaria, scarlet fever, typhoid, measles, sepsis.
- [3. *Serum Disease*.]
4. *Primary Local Affections of Stomach and Intestinal Mucous Membrane*:
 - (a) Corrosive toxic gastritis.—Ingestion of vegetable poisons, metal salts, acids, and brine.
 - (b) (Gastric and) intestinal ulcers.—[Typhoid,] tuberculosis, dysentery, influenza, follicular enteritis after measles, membranous enteritis, enteroliths, [round ulcer,] intestinal parasites (anchoylostomum).
 - (c) Intussusception (At first feces with blood and well preserved intestinal epithelium in large pieces; later,

B. IN OLDER CHILDREN.—Continued

pure blood; with symptoms of intestinal obstruction, *q.v.*).

- (d) **Hæmorrhoids** (in constipation), polypi of the rectal mucous membrane (Unchanged blood in every stool, digital examination discovers a soft pediculated tumor only a few centimetres above the anus, especially in children who have had intestinal disorders).—Injuries to the mucous membrane through enemas, sexual abuse, digital examination, in anal fissure, condyloma, prolapse of the rectum.

5. Acute Yellow Atrophy of the Liver, Syphilitic Hepatitis. Cirrhoses accompanied by Icterus.

45. SYNDROME OF (PARTIAL OR COMPLETE) INTESTINAL OBSTRUCTION

Abdominal pain, obstinate constipation and absence of flatus, uncontrollable vomiting (first chyme, then bile, blood, and possibly fecal matter), meteorism; ineffectual visible peristalsis, intestinal rigidity, collapse.

	Obstruction in upper small intestine.	Obstruction in colon (lower half).
Meteorism	Changing, moderate, and only epigastric; lower and lateral parts of abdomen sunk in.	Constant, strong, and universal; fecal masses palpable.
Vomiting	None, or only occasional. Fecal.	Fecal.
Urine	Indicanuria, oliguria.	No indicanuria and oliguria.
Collapse	Of the gravest kind.	Moderate.

May be mistaken for acute intestinal colic, acute intoxications [twisting of movable kidney].

A. IN NEWBORN CHILDREN.

Congenital malformation after foetal pathological conditions or arrest of development; *e.g.*, atresia ani, recti, jejuni, duodeni, pylori (Grave state, debilitation, subnormal temperature, dyspnoea, convulsions, visible changes at the anus, palpable changes in the rectum). Congenital umbilical hernia. Persistent ductus omphalomesentericus.

B. PREDOMINATING IN NURSINGS.

Intussusception (acute ileocecal). After traumas, in polypi, partial paralyses, relaxation of the mesentery through loss of fat (Sudden onset, vomiting generally the initial symptom; sausage-shaped, movable tumor which encircles the umbilicus to the left; circumscribed pain on pressure; bloody stool—often pure blood as early symptom—later prolapse and meteorism. Intestinal obstruction plus bloody stool is pathognomonic in nurslings).

C. PREDOMINATING IN OLDER CHILDREN.

1. Appendicitis (make rectal examination) (Tumor only slightly movable, cannot be grasped all round, reaches down into the pelvis; pain on pressure at McBurney's point; no bloody stool!; temperature and pulse!; abdominal wall stiff like a cuirass). *See also Appendicitis, No. 51.*
2. Peritonitis with paralytic ileus (Distention of the abdomen, fever, pain; no visible peristalsis; only exceptionally fecal vomiting).
3. Foreign bodies in the intestine; *e.g.*, indigestible food remnants, especially fruit kernels, scybala, fecal stones, ascaridic balls, tænia (Seldom causing complete occlusion; finding on palpation!; pain; often bloody stools and bloody vomit, eructation; perhaps signs of beginning perforation).
- [4. Incarcerated hernia (inguinal, umbilical,) (findings at the mouth of the hernial sac!), extreme distention of bladder, cicatrization after ulcerous processes (dysentery, tuberculosis, syphilis), tumors (sarcoma, enlargement of lymph-glands), colicky pains, ligation through peritonic cords, volvulus.]

46. INCONTINENCE OF FECES

A healthy, well nursed child learns to avoid defecation in bed- and body-linen in the course of his second year, possibly only toward the end. Later, involuntary evacuation of feces, frequently only of fluid stools, during fright or terror.

1. Disorders of innervation (sphincter paralyses) in organic affections of the nervous system.—Tumor cerebri (especially lesions of the crus cerebri), meningitis III; tumors, syphilis, and hæmorrhages of the spinal cord, transverse myelitis, juvenile ataxia, spina bifida, tabes; polyneuritis, post-diphtheritic and post-typhoid paralysis [anterior poliomyelitis]; functional neuroses (chorea minor).

2. Disturbance of consciousness, and epileptic seizures.—Epilepsy (sporadic occurrence at certain intervals; even before the actual paroxysms happen), eclampsia, laryngospasms, pathologic deep sleep, cerebral pneumonia.

3. Psychic defects, idiocy, degeneration; hysteria. Also, educational defects.

4. Extreme bodily debility, state of exhaustion.—Collapse, agony; myocarditis after acute infectious disease.

5. Sphincter lesions of a traumatic nature.—Sexual abuse.

47. TENESMUS AND PAIN IN DEFECACTION

1. Intestinal affections (often with bloody and purulent stools: dysenteriform picture), appendicitis (pelvic), mucomembranous enteritis, invagination, intestinal tuberculosis, [typhoid,] dysentery, dysenteriform enteritis in uræmia, metallic poisoning (Hg, As), after infectious diseases (measles and serum injection; intestinal parasites; fissure of anus, periproctitic abscesses, syphilitic fissures and condylomata, polypus of the rectum (hæmorrhoids); foreign bodies in the terminal portion of the intestine.

2. Peritoneal affections.—Peritonitis.

3. Genito-urinary affections.—Acute nephritis, [urinary calculus (only in erect position, accompanied by pain in the gastric region)].

48. CHANGES OF THE ANUS

A. TUMOR, protruding either continually or periodically.

1. *Prolapse of Rectal Mucosa*, Prolapse of Anus and Rectum (Cylindrical sausage-shaped mass, bleeding easily; readily replaceable; more or less direct transition of the exterior covering of the tumor into the anal integument; "fornix" never very deep; no sign of intestinal obstruction).

- (a) In inflammatory and other affections of rectal mucous membrane (colitis, dysentery, diphtheria, polypi).
- (b) In genuine paralysis of the sphincter (affections of the spinal cord, myelocoele, spina bifida, leptomenigitis).
- (c) In relaxation of the sphincter through overextension in pressing and straining (constipation, phimosis, urinary calculi, hæmorrhoids, oxyuris, whooping-cough), because of general debility.

2. *Prolapsed Ileocecal Intussusception* (Grave picture of intestinal obstruction (*q.v.*) of 3 to 5 days' duration; 'deep "fornix"; central aperture; possible existence of appendicitis).

3. *Polypus of Rectal Mucosa*, prolapsed (Tumor small, roundish, very movable, pediculated, slightly painful, and frequently cyanotic, without central opening, and generally bleeding profusely; no severe general symptoms. At times protruding only just beyond the sphincter; palpable. Sometimes multiple).

[4. *Hæmorrhoids* in "arthritis," in hereditary disposition, blood congestive factors and local irritations (Generally multiple, blue or violet, painful, hot, erectile, and compressible papillæ, and lobules, situated partly below, partly above the sphincter).]

B. OTHER CHANGES.

1. *Fissures, Ulcers* (Hard stool, contractions of sphincter, hæmorrhage and pain in defecation).—Congenital and acquired syphilis, dysentery, tuberculosis, gonorrhœa, and non-specific conditions, such as intertrigo, etc.
2. *Fistulæ after Perianal and Periproctitic Abscesses* (Pains and continuous secretion).
3. *Papulæ* (syphilitic) and *Membranous Coating* (diphtheritic), *Ulcers* (tuberculosis), *Pruritus and Effects of Scratching* (oxyuris, hemorrhoids).—Intertrigo (thrush and intestinal conditions. Congenital anal and anorectal atresia with or without communications (vagina, bladder, urethra).

49. ABNORMAL CONDITIONS IN RECTAL EXAMINATION BY PALPATION

First evacuate the colon. Patient in left lateral position with hips and knees flexed. The physician, standing at the right, introduces his short-nailed index finger, well cleansed and greased*, gradually, carefully, and systematically into the anus, up the intestinal tract as far as possible, palpates the intestinal contents, and its outer surroundings through the walls of the intestine, always comparing right and left, the left hand assisting at the abdomen. Attention should also be paid to pain on pressure.

TUMOR EXAMINATION.

Simulated by full bladder.

Rectal polypus	}	(Tumor situated in lumen of intestine; can be grasped all round. Peritonitic and ascitic effusion (rather considerable) into the abdomen, demonstrated by cystic, fluctuating, lateral bulging of the rectal wall).
Intussusception		

Appendicular abscess (Roundish or grapelike resistance, painful, adherent to right pelvic side, from there protruding nearly to median line).

Periproctitic abscess.

Tumors of the lymphatic glands, especially tuberculosis (Large knots and bundles).

[Hæmorrhoids situated high up; salpingitis, gonorrhœal oophoritis in vulvovaginitis; true tumors of various organs of the true pelvis; formation of urinary calculi.]

ROUGHNESS OF THE PERITONEUM in Douglas's pouch.—Tuberculous peritonitis.

50. PATHOLOGICAL PICTURE OF TYPHOID FEVER

Typhoid fever is especially simulated by a series of conditions which may have the following signs (together with the presence of pathogenic

*A rubber finger cot should always be employed.

germs in the blood) in common with typhoid: Status typhosus (prostration, stupor, delirium, fever, often excessive); pains in the head, limbs, and abdomen, acute infectious splenic tumor; roseola and kindred skin eruptions; hæmorrhages of skin and mucous membranes; diazo reaction of the urine.

The Following Favor Typhoid: Fuliginous tongue, continued high fever in the beginning), typical roseola, leukopenia, relative bradycardia, characteristic stools, bronchitis. Gruber-Widal reaction*).

The Following do not Favor Typhoid.—Sudden onset, subsiding of local symptoms, constipation, icterus, Kernig's sign; absence of status typhosus (in younger children), roseola, diazo reaction, and Gruber-Widal in the first week of the disease.

The Following Speak against Typhoid.—Strong perspiration, coryza, leucocytosis, herpes, scaphoid belly; absence of splenic tumor, diazo reaction, and of Gruber-Widal in the second week.

1. Acute miliary tuberculosis (Steep, irregular oscillations of temperature; generally no distinct stupor; no diarrhœa; general hyperæsthesia; rapid emaciation; tachycardia; dyspnœa and cyanosis with no pronounced pulmonary signs; moist tongue; hydriatic measures have no manifest effect upon mind and general condition).

2. Acute ulcerative endocarditis (with emboli) (Usually sudden onset; cardiac pain, later cardiac murmur of variable nature; chills and fevers; diarrhœa; erythema or purpura).

3. Cryptogenetic septicæmia and pyæmia with hæmorrhagic diathesis, sinus-thrombosis, etc. (Articular affections, pus fever, icterus, violent osseous pains, tachycardia, skin exanthemata, and hæmorrhages).

4. Osteomyelitis acuta (Local symptoms! Leucocytosis).

[5. Putrid typhoid, and Weil's disease (Rapid and favorable course; Gruber-Widal reaction may be present; sometimes icterus).]

[6. Malaria, in the rarer forms (Quinine acts).]

7. Malignant septic measles (Catarrhal syndrome, exanthema!).

8. Scarlatina typhoides (Angina, exanthema, usually sudden onset).

9. Croupous pneumonia, with late exhibition of local symptoms (Chills and fever, type of respiration!, pain on deep inspiration, herpes, subicterus, disappearance of the patellar reflex, acetonuria, leucocytosis, great coagulability of the blood).

10. Rheumatic polyarthritis (atypical course) (Local manifestations

*Technic of the Gruber-Widal reaction.—An emulsion of a 24-hour agar culture of typhoid bacilli in bouillon (in case of need, also dead culture: Ficker's reaction), is mixed in different specimens with different quantities of serum, or else with the blood of the patient, and the mixture is observed both macroscopically and microscopically (in hanging drops) for about 2 hours after mixing 95° F. (35° C.). Clearing and sedimentation (macroscopic in a solution of serum of at least 1 : 50) is proof of existing (or previously existing) typhoid. Procedure in taking the blood samples and production of the series of solutions is accomplished quickly, safely, and delicately by the serum mixer (*Münchener medizinische Wochenschrift*, 1905, No. 7).

in joints, sporadic involvement of some joints by superficial, painful exudations; salicylic acid has effect!; strong, sour perspiration, sudamina).

Add to this:

11. "Influenza gastrica" (Sudden access of fever, initial fever, coryza, grayish white coat of the tongue, headache, total distaste for food in case of diarrhœa, colicky stools).

12. Acute gastro-enteritis, indigestion with fever (Usually no splenic tumor, pronounced dyspeptic manifestations, colicky pains, mucous stools, herpes labialis).

13. Acute peritonitis (Severe pains, continuous vomiting, effusion into the abdominal cavity, leucocytosis).

14. Ascending cystopyelitis.

15. Meningitis (*see No. 180*).

16. Autotoxic processes, such as intestinal ephemeral fever with acetonæmia and uræmia (Continuous albuminuria under usually superficial cerebral irritative manifestations; no bradycardia; no typhoid fever).

[17. Botulism, intestinal parasites, Hodgkin's disease.]

NOTE.--In the typhoid of small infants there are frequently no other symptoms of disease than continuous or remittent fever, mucous or hard stools, ill temper, and loss of appetite.

51. DISORDERS SIMULATING APPENDICITIS

High fever, uncontrollable vomiting, pains in the right iliac fossa, œdema at the same place, dulness, muscular rigidity, collapse, singultus.

1. *True Appendicitis* (Circumscribed pain on pressure at the McBurney point*; frequently sudden, spontaneous pain around the umbilicus or in the right side of the stomach; hyperæsthesia of the integument at that spot; marked resistance; cuirasslike, at the beginning almost scaphoid belly; tumor in the fossa iliaca dextra, characteristic result of anal palpation; light dermatic œdema over Poupart's ligament; facies abdominalis; tachycardia with normal or only slightly raised temperature; leucocytosis of over 20,000, with suppuration!; dysuria. Not against appendicitis is a diarrhœa from the beginning, or position of the demonstrable pathological focus outside the typical spot).

2. *Other Inflammatory and Ulcerative Gastro-Intestinal Processes:*

(a) Typhoid fever (commencing atypically) (Pain less violent and less sudden in onset, more diffuse; less muscular resistance and cutaneous hyperæsthesia; diazo reaction. Paratyphoid appendicitis may follow!).

(b) Colitis and dysentery (Descending colon the most painful; resistance and susceptibility to pressure of the skin less distinct; characteristic stools. Appendicitis may follow!).

* McBurney's point is situated between the lateral and median third of the distance between umbilicus and right anterior superior spine of ilium.

2. *Other Inflammatory and Ulcerative Gastro-Intestinal Processes :*
—*Continued*

- (c) Enteritis after indigestion (Pain not at McBurney's point, and only of short duration; less severe picture; no collapse). Gastric fever, membranous enteritis.

[(d) Ulcer of the stomach (Hæmatemesis, bloody stool).]

- (e) Intestinal tuberculosis.

3. *Other Conditions of Intestinal Obstruction (see also No. 45).*

- (a) Invagination (In younger children, tumors differently localized, more superficially situated, cannot be grasped all round; early bloody stool).

- (b) Typhlitis stercoralis (No distinct finding at McBurney's point; less sudden onset; usually only vague pains along the entire colon, the latter distended from the first; no resistance, and no vomiting).

[(c) Hernia incarcerata.]

4. *Acute Peritonitis of Other Origin:*

- (a) Acute tuberculous peritonitis (Antecedents, pulmonary and glandular manifestations, ascites).

- (b) Gonorrhœal and pneumococcal peritonitis (No pronounced finding at McBurney's point, no dulness, little tension of abdominal walls; diarrhœa, primary foci in the organism).

- (c) Perforative peritonitis (*e.g.*, starting from Meckel's diverticulum, etc.).

From this source also the peritonitic irritation (enanthema?) in prodromata of measles.

5. *Certain Acute Infectious Diseases* with abdominal symptoms in the initial stage:

- (a) Croupous pneumonia ("pseudo-appendicitis pneumonica") (No resistance of the abdominal walls; pains superficial).

- (b) Measles.

6. *Certain Liver Affections* in other intestinal processes:

- (a) Acute congestion and degeneration of the liver (Great pain below the lower ribs on the right, considerable hepatic tumor, muscular resistance only in the upper parts of the abdomen).

[(b) Cholecystitis.]

7. *Genito-Urinary Affections:*

- (a) Pyelitis (Pain more medial; examination of urine!).

[(b) Floating kidney, renal colic.]

8. *Various Abscess Formation* in the abdominal region.—[Prostatitis, ulcerations of the adnexa, psoas abscesses (few peritoneal manifestations, typical position of legs), perinephritic abscesses and abscesses of the abdominal muscles).]

9. *Intoxications* (lead, acetonæmia).

10. *Hysteria* (Can imitate almost all signs; general picture however usually lighter, hardly misleading; examination of blood, rectal palpation!).

[11. *Purpura* (Henoch).]

52. DISTENTION OF THE ABDOMEN

Normally the anterior abdominal surface lies on a level with the anterior thorax wall when the patient is lying on the back.

1. *Distention of Stomach and Intestine with Gas (Acute)*: meteorism, chronic, habitual, "tympanites" (Aircushion feeling, tympanitic sound, displacement or superposition of diaphragm and liver).

(a) On account of abnormally large formation of intestinal gases.—Dyspepsia, chronic gastric and intestinal catarrh, isochymia, marasmus (carbohydrates!), overloading of stomach.

(b) On account of increased resistance to expulsion.—Obstruction of pylorus (distention of stomach with collapse of the rest of the abdomen), obstruction of intestine (*q.v.*, No. 45), congenital dilatation of colon and congenital megacolon (distention and difficult defecation, often from birth), glandular tuberculosis, peritoneal bands.

(c) On account of diminished power of expulsion.—Habitual gastric and intestinal atony with dilatation (in rachitis, etc.), intestinal paralysis (peritonitic, toxic, and in acute infectious diseases: typhoid, pneumonia, miliary tuberculosis).

(d) In various other conditions: Congenital syphilis and "parasymphilis," myxœdema, cretinism, hysteria, purpura hæmorrhagica and melæna, intestinal parasites.

2. *Loading of Stomach and Intestine with Fecal Masses*.—Constipation.

3. *Enlargement of Abdominal Glands* (solid tumors), *Enlargement of the Liver* through (post-infectious) cardiac weakness, enlarged spleen (*q.v.*).

4. *Effusion of Fluid into the Abdominal Cavity* (Ranular abdomen, lateral portions hanging down in the recumbent position, pointed abdomen in the erect position; more or less movable; dulness; fluctuation).

5. *Paralysis of the Abdominal Muscles* (spinal and peripheral conditions).

53. SINKING OF THE ABDOMEN

1. Empty intestine.—Insufficient food supply (stenosis of pylorus and œsophagus, habitual vomiting, cachexia); after diarrhœa.

2. Contraction of the intestine.—Colitis, dysentery, enteritis, lead colic, hysteria, appendicitis, invagination.

3. Contraction of the abdominal walls (and the intestine).—Meningitis (cerebrospinal, especially basilar) (Spastic, hard abdominal walls, scaphoid belly, unless exudative peritonitis is likewise present).

[4. Also in diaphragmatic hernia, heat stroke, etc.]

54. VISIBLE PERISTALSIS

Frequently increased through irritation by warmth or cold at the abdominal integument.

In abnormally thin or relaxed abdominal walls (atrophy; central paralysis; *e.g.*, in meningitis), normal peristalsis is sometimes visible.

Increased peristalsis of the stomach in pyloric stenosis (organic and spastic), and of the intestine in overfeeding and in intestinal obstruction, and stenosis in congenital dilatation of the colon and strong intestinal irritation (acute inflammatory processes).

55. COLICKY PAINS

Abdominal or colicky pains are often complained of by children, when the pain or discomfort is due to other causes.

A. PAIN IN THE ABDOMINAL WALLS.

1. *Hyperæsthesia of the Abdominal Integument* (pseudoperitonitis) (Frequently also other painful zones than abdominal).—Hysteria, typhoid, meningitis, appendicitis, croupous pneumonia.
2. *Pain in Muscles and Aponeuroses*.—Polymyositis, gymnastic overexertion, spastic cough; straining in dysuria, ischuria, urolithiasis.
3. *Radiating Pains from Other Regions*.—Incipient pneumonia, pleuritis (not increased upon pressure), nephritic processes.
4. *Neuralgia of the Abdominal Walls*.
5. *Incarcerated Ventral Hernia in Separation of the Recti Muscles*.

B. PAIN IN THE ABDOMINAL ORGANS.

1. *Gastralgia, Cardialgia*:
 - (a) Chlorosis (Bulging of the epigastrium, dyspeptic and atonic conditions with cardiospasm; irritable weakness of the nervous system).
 - (b) Chronic dyspepsia with atony of the stomach; pyloric stenosis; indigestion, intoxication.
2. *Enteralgia, Intestinal Colic* (Generally on forced intestinal movement), in the latter case rather improved by pressure, also after evacuation of stool and vomiting; often without fever; distinctly paroxysmal):

B. PAIN IN THE ABDOMINAL ORGANS.—Continued

- (a) Inflammatory (stomach and) intestinal processes.—Gastritis, enteritis of all kinds, especially colitis, dysentery, typhoid (ileocecal region, splenic or umbilical region, usually in the later course, not very severe), tuberculosis.
- (b) Stenotic intestinal conditions.—Congenital stenosis, invagination (usually in the right iliac fossa, occurring suddenly and continuously, invagination of small intestine less painful than the more frequent ileocecal form). Appendicitis (McBurney's point, frequently setting in quite suddenly, often radiating as far as the umbilicus). Impaction of feces (indicanuria, fecal tumors, therapy!). Intestinal parasites, especially ascarides (stabbing, boring, intermittent pain, usually around the umbilicus; salty and acid food increases, fatty food mitigates). Other kinds of intestinal obstruction (*q.v.*).
- (c) Catching cold.
- [(d) Intoxications, especially lead,] Henoch's purpura.
- 3. *Peritonitic Pain* (Violent, permanent, increasing on pressure; at first in the umbilical region, later diffused; tense abdominal walls, disappearance of the liver dullness, etc.).—Acute (and chronic) peritonitis; "peritonism" in measles (enanthesma?) (crepitation and friction); gonorrhoeal vulvovaginitis, etc.
- 4. *Pain in Kidney* (radiating toward lumbar region, also toward the bladder).—Nephritis [movable kidney, nephrolithiasis, and other renal processes]. *See also Lumbar Pain.*
- 5. *Pain in the Liver*.—Especially pericarditic cirrhosis.
- 6. *Pain of the Lymphatic Glands*.—Tuberculosis (around the umbilicus).
- 7. *Ovarian Pains*.—Ovaria; hysteria.
- 8. *Undetermined Pains* in the abdomen in acute infections, initially (pneumonia, influenza), and especially in later cardiac insufficiency (diphtheria, scarlet fever).

56. EFFUSION INTO THE ABDOMINAL CAVITY

Fluid Exudate or Transudate into the free peritoneal cavity.

Fluctuation (possibly distinctly palpable from the abdominal ring), more or less movable dullness from time to time, in the portions affected, distention of the abdomen; obliteration of the umbilicus; lustre and greater tension of the abdominal integument traversed by dilated veins. Elevation of the diaphragm, disappearance of the liver dullness.

Demonstration: (As little as 150 to 200 c.c. are demonstrable; 800 c.c. produce visible deformity.) Fluctuation test, movability test, determination of the limits of dulness; all this after emptying both bladder and intestine. Puncture between mammillary and axillary lines below the umbilical level in the region of dulness, always with a Mandrin cannula, the lumen of which should not be too small (1-2 mm.).

May be simulated by meteorism, myxœdema, great abundance of fat in the abdominal wall (false fluctuation prevented by interposed finger), great distention of bladder, large cystic tumors, hydronephrosis.

Character of the fluid obtained by puncture:

	Appearance.	Color.	Specific gravity.	Albuminous contents.	Spontaneous coagulability.	Sediment.
Transudate...	Transparent.	Light yellowish.	D < 1.015; usually D < 1.012.	A < 1-2 (-4) per cent.	Absent.	Very few leucocytes and endothelia; no germs.
Exudate* (serofibrinous).	Usually cloudy (like buttermilk).	Greenish yellow.	D > 1.015.	A > (3-) 4 (-6) per cent. (the higher the percentage, the fresher the process).	Present.	Many leucocytes and endothelia; often blood corpuscles and pathogenic germs.

[Chylous and chyloform effusions (fluid milkily clouded, containing the finest albuminoid granules and fat) are very rare in infancy; purulent and seropurulent effusions are met with in perforative processes.]

In exudation, there usually exist, in conjunction with the general signs of the effusion, pains, collapse, fever, singultus, vomiting, dysuria, tachycardia, tachypnœa, constipation, splenic tumor, and frequently friction in the region of the liver.

In transudations, these signs are usually wanting; there exists a stronger net of dilated collateral veins on the anterior abdominal wall. The mobility of the dulness, through change of position, is usually very distinct, in comparison with that found in exudation.

A. TRANSUDATIVE EFFUSION (HYDROPS ASCITES).

1. *Disturbance of Cardiac Function.*—Insufficiency of cardiac valves, pericarditis, myocarditis. Cardiac insufficiency in chronic pulmonary affections (œdema, dyspnœa, and cyanosis preceding, objective signs of the cardiac and pulmonary affections; albuminuria in oliguria).

2. *Pathological Conditions of Abdominal Organs* (congestion of the portal system):

(a) Hepatic diseases, cirrhosis, degeneration, genuine tumors (Considerable effusion, rapidly recurring after

* Exudates which have been kept for some time occasionally assume the character of transudates; on the other hand, an exudative process may make its appearance beside the existing transudate. Under some circumstances sedimentation occurs within the body.

A. TRANSUDATIVE EFFUSION (HYDROPS ASCITES).—Continued

evacuation; strong venous net, always splenic tumor, hepatic changes, functional hepatic insufficiency; urobilinuria, icterus, alimentary glycosuria, epistaxis, cachexia).

(b) Diseases of the pancreas and renal adnexa (Pancreatic symptoms, bronze skin).

(c) Tumors of the mesenteric glands in tuberculosis and pseudoleukæmia [mesenteric tumors].

3. *Diseases of the Kidneys, Hydræmia.*—Acute and chronic inflammations, degeneration; tumors (Anasarca, continuous albuminuria, cylindruria, uræmia, pallor).

4. *Grave Anæmias and Cachexia.*—After typhoid, scarlet fever, measles; leukæmia, pernicious anæmia (Slow occurrence of a slight effusion; cachexia; history, blood examination).

B. EXUDATIVE EFFUSION (PERITONITIS).**1. Chronic Peritonitis:**

(a) Chronic serofibrinous peritonitis.—In the course of inflammatory processes of the abdominal organs (intestine, spleen, liver) after traumatism, colds(?), burns; in beginning ovulation (Almost exclusively in childhood and puberty; long stationary condition, duration 3 months at the most, then relapse; constipation, no icterus, no splenic tumor, no fever no pseudotumors in the abdomen; exudate free, never hæmorrhagic, contains polynuclear leucocytes).

(b) Chronic tuberculous peritonitis (ascitic form) (Oval shape of abdomen, protrusion especially below and around the umbilicus; œdema of umbilical integument; diarrhœa, pain, pseudotumors, fever, emaciation; meteorism sometimes hides the exudation. Course protracted and obstinate; often transition into the fibrous and caseous form; frequently accompanied by pleurisy. Exudate encysted, often contains blood and many lymphocytes).

2. *Acute and Subacute Peritonitis* (Usually sudden onset with high fever, severe pains, diarrhœa, "facies abdominalis," tachycardia; later on meteorism, dysuria):

(a) *Acute perforative peritonitis.*—After the formation of ulcers (tuberculosis, dysentery, typhoid, appendicitis); after opening of abscesses (perityphlitis, perinephritis, peripleuritis); after gangrene (incarcerated hernia, invagination, congenital intestinal occlusion).

B. EXUDATIVE EFFUSION (PERITONITIS).—Continued

- (b) Acute peritonitis through extension of inflammatory processes of the abdominal organs (liver, stomach, intestine, umbilicus, kidney) and thoracic organs (especially pleura). Also in ascending gonorrhœal vulvovaginitis (Often severe general picture with sudden onset, simulating appendicitis, but without local findings at the McBurney point, and usually taking a favorable course).

- (c) Acute peritonitis through invasion of pathogenic germs by way of the circulation of the blood from diseased organs (metastatically), streptococcus sepsis, sepsis in scarlet fever, erysipelas, and rheumatism.

Peritonitis (neonatorum) in streptococcus sepsis (Chiefly in the first week of life; suppurative fluid exudation, often recognizable by the still open tunica vaginalis of the scrotum, no encapsulation; simultaneously icterus, umbilical ulceration, hæmorrhages, articular inflammations; rapid, fatal course, in a few days; demonstration of streptococci!).

Pneumococcus peritonitis of older children in preëxisting pneumonia or after intestinal processes (Sudden onset with pains, vomiting, diarrhœa, distention, fluctuation; profuse, ulcerous effusion; frequently encysted and taking a favorable course; more rarely universal and fatal).

Polyserositic peritonitis of nurslings (Heubner), together with encysted ulcerous pleuritis (Usually fatal).

[Fœtal peritonitis, traumatic peritonitis.]

57. TUMORS AND PSEUDOTUMORS IN THE ABDOMINAL REGION

(Abnormal, circumscribed resistance and dulness, exclusive of tumors of liver and spleen)

Demonstration: First examine in the dorsal position, best with the mouth open and hips and knees flexed. The child's attention should be distracted; deep palpation should be insinuatingly accomplished (with warm hands!); bimanual procedure, one hand pushing the tumor from the rectum, the lumbar region, from the sides, above or below, to play into the other hand. Examine behavior in change of position. In some cases anæsthesia is very useful. Radiography.

A. PERTAINING TO THE ABDOMINAL WALLS (Superficial situation; movable with the abdominal walls).

1. Stiffening of the abdominal walls (muscular rigidity) in appendicitis, peritonitis, and hysteria (Often as hard as a board, cuirass-like).

A. PERTAINING TO THE ABDOMINAL WALLS, etc.—*Continued*

2. Hæmatoma and abscess of the abdominal walls (of the recti muscles in typhoid, after traumatism, after appendicitis) (With hard, sharp border, over which the skin is mobile; hæmorrhage usually absorbed spontaneously).

B. PERTAINING TO STOMACH AND INTESTINE (Accompanied by constipation and intestinal occlusion).

1. Stiffened and contracted portions of the alimentary tube.—
“Phantom tumors” (paralytic tympany and contraction of abdominal muscles) (Not painful, no dulness; disappears during sleep, warm bath, and narcosis). Contracted rectum and colon (Rubber-tube-like growth in the depth of the left hypogastrium). Stiffening of intestine before intestinal stenosis, pylorospasm (Syndrome of intermittent benign pyloric stenosis; tumor rarely palpable).
2. (Acute Ileocecal) Intussusception (Sudden onset, at first afebrile; intestinal obstruction; manifestations of peritoneal irritation almost always serious; bloody stools; hard tumor, sausage- or horseshoe-shaped, in the left—rarely right—mesogastrium, encircling the umbilicus, with frequent spontaneous changes of shape, indurated; the intussusceptum may often be reached as tumor through the rectum, or it may appear at the anus).
3. Appendicitis, infiltrated appendix, empyema, appendicitic and paratyphlitic exudate (Usually at McBurney's point or nearer the iliac crest, ridgelike, painful; intestinal obstruction).
4. Impacted feces, scybala, engorgement of the intestinal contents previous to each obstruction (Roundish, very mobile, doughy, plastic, especially in the hypochondria, disappear after laxatives).
- [5. Worms (ascarides).]
6. Hyperplasia [and genuine tumors]. Hypertrophy of the pylorus (Syndrome of the grave organic pyloric stenosis occurring at the latest in the second month of life; tumor hazelnut-shaped, at the right of the median line, near the transverse fissure of the liver; peristalsis of the stomach, uncontrollable vomiting).
- [7. Carcinoma and sarcoma of stomach and intestine.]

C. PERTAINING TO THE PERITONEUM.

1. Circumscribed thickening of the peritoneum and the omentum, inflammatory cords, callous thickening of the intestinal serosa, adherent intestinal convolutions (Especially in the region of colon and umbilicus, cords travelling trans-

C. PERTAINING TO THE PERITONEUM.—*Continued*

versely or obliquely through the abdomen—nodular, indurated, and often painful on pressure). Encysted peritoneal abscesses (subphrenic, appendicular, cold abscess after spondylitis) (Prevertebral, fluctuating).

[2. Sarcoma, carcinoma, peritoneal cysts.]

D. PERTAINING TO THE KIDNEYS (Intestine always superposed, not mobile with respiration; with functional derangements of the kidney, usually change of the urine; *see also Nephritic Tumors*).

1. Movable kidney (In girls; with colicky pains; tumor is movable in the position of the normal kidney).

2. Hydronephrosis (Tumor situated by the side of the spinal column; not very painful on pressure).

3. Perinephritic abscess (Tumor in the lumbar region, spinal column very painful; stiff, legs drawn up).

[4. Nephritic sarcoma and carcinoma (Usually under 5 years of age).]

E. PERTAINING TO OTHER ABDOMINAL ORGANS.

Bladder greatly distended, tumors of the mesenteric glands (status thymicus, infectious intestinal conditions, pseudoleukæmia, tuberculosis) (At the level of the umbilicus, in spinal region, slightly mobile, indurated, somewhat painful on pressure; rarely palpable with distinctness; most frequent form of swelling in the abdomen).

58. HEPATIC "TUMORS"

Often recognizable by the bulging caused in the abdominal wall and the right costal arch by an organic mass movable with respiration.

Demonstration by palpation (and percussion).

Physiologic limitation of the superficial hepatic dulness, with considerable individual variations; average measurements (in dorsal position) according to illustration on opposite page, in centimetres:

	b_1	b_2	b_3	a_1	a_2	c
Newborn.....	3.0	3.9	...	...	2.0	2.6
Nursling.....	3.6	4.4	3.5	2.7	1.5	3.2
Infancy.....	4.8	5.7	4.2	3.7	1.1	3.8
Childhood.....	5.8	6.9	5.3	4.2	0.9	4.0
Puberty.....	6.7	7.8	6.3	...	0.9	5.2

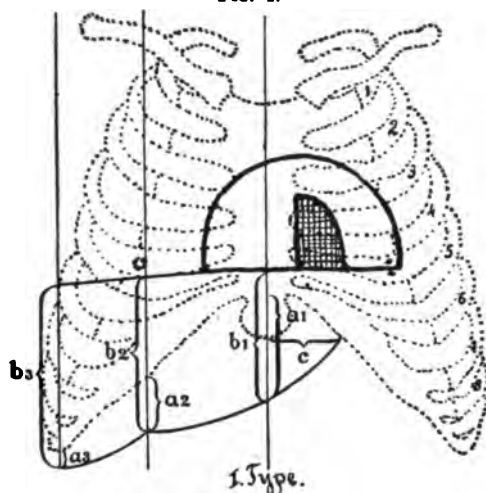
The upper border of hepatic dulness lies in the right mammillar line at the fifth to the sixth rib and runs nearly horizontal.

Simulated by displacement and pressing forward of the left lobe (pleural exudate, emphysema, subphrenic abscess, pneumothorax), by perihepatic appendicitis, and by floating liver.

A. STOOLS ACHOLIC, CHOLÆMIA.—Icterus from biliary obstruction (in older children) (liver slightly enlarged, somewhat sensitive to press-

ure, smooth surface, slight resistance; seldom before 3 years of age, almost never under 1 year; beginning acute, duration generally not more than 3 weeks; see *Icterus*). [Icterus from biliary obstruction in newborn children consequent on obliteration of the bile ducts, see *Icterus*.]

FIG. 1.



B. STOOLS NOT ACHOLIC, or only temporarily acholic.

1. *Hyperæmia of the Liver* (Subicterus, may also be absent):

- (a) Active, in the beginning of acute infectious diseases (especially typhoid, scarlet fever, measles, pneumonia, diphtheria, sepsis neonatorum, Weil's disease) (Liver only slightly enlarged and painful on pressure, smooth, non-indurated, tumor always transitory).
- (b) Passive, in cardiac and pulmonary affections (passively congested liver); also degeneration of heart muscle in infectious diseases (diphtheria) (Liver sometimes considerably enlarged, pulsating, tender, harder; tumor constant, at times increasing; size changes, dependent on the cardiac force; generally no ascites, no enlarged spleen; causative affection).

2. *Degeneration of Liver* (Slight or no icterus, in the urine more or less distinct signs of functional hepatic insufficiency; diminution of urea, alimentary glycosuria, urobilinuria; tendency to hæmorrhages, œdema, and diarrhœa):

- (a) Fatty degeneration, infiltration, and cloudy swelling.
—In intoxications (phosphorus, alcohol, santolin,

B. STOOLS NOT ACHOLIC, etc.—Continued

etc.), in infectious diseases (as above, besides miliary tuberculosis and gastro-enteritis of nurslings) (Liver often enlarged, soft, smooth surface, sharp border, moderate pain on pressure; condition stationary for a long time, progressive in general; no icterus, no ascites, no collateral circulation, no enlarged spleen).

- (b) Amyloid degeneration in tuberculosis, syphilis (especially if causing chronic ulcerations), rachitis (?) (Liver very large, very hard, edges rounded off, surface smooth; little pain on pressure; rarely icterus; functional insufficiency occurring only later, diarrhoea, enlarged spleen, albuminuria, usually no ascites, no strong collateral circulation; condition stationary for a long time).

3. *Abscess of the Liver*.—Pyæmia, phlebitis umbilicalis, traumatism, immigration of ascarides, appendicitis and other abdominal ulcerative processes, typhoid, tuberculosis, seldom dysentery (Liver generally enlarged *in toto*, painful; also (not always and often only at a late stage) a circumscribed bulging of the abdominal wall, with œdema of the skin and venous plexus; remitting fever, chills and fever, usually icterus, hæmorrhages, typhoid and meningeal manifestations, generally no ascites, no enlarged spleen; pains radiating toward the shoulder; puncture may yield pus).

4. *Cirrhosis* (Hepatitis) (Infantilism, itching, dry skin, functional hepatic insufficiency, swelling of terminal phalanges (osteoarthropathy), varying disturbances of digestion).

- (a) Syphilitic cirrhosis (interstitial or gummatous; the former congenital, the latter usually developing in the first year of life) (Liver at times enormously enlarged, greatly protruding, indurated, sometimes irregularly shaped, with retractions and tuberosities; marked venous plexus, much enlarged spleen; anasarca and ascites may be present to a considerable extent; icterus rare; pain on pressure; no biliary pigment in urine, indicanuria).

- (b) Cardiac and "cardiotuberculous" cirrhosis (especially in adhesive tuberculous pericarditis and rheumatic serositis, together with recurring pleuritis and tuberculous peritonitis) (Liver often greatly enlarged,

B. STOOLS NOT ACHOLIC, etc.—*Continued*

volume dependent on the condition of the heart for the time being; venous plexus, splenic tumor, ascites; icterus absent or slight; pain!; functional hepatic insufficiency; œdema of the abdominal integument and the lower extremities; cachectic hæmorrhages; dyspnœa and cyanosis; manifestations in the heart itself may be absent, congestion of the general circulation).

(c) Biliary cirrhosis (among other diseases, in hereditary syphilis—here possibly accompanied by icterus from obstruction of the bile) (Liver smooth, evenly large, and hard, no venous plexus, splenic tumor large and indurated, ascites almost always absent, icterus early and very pronounced, bile pigment in the urine, functional hepatic insufficiency, fever, pain, leucocytosis, hæmorrhages, very chronic course).

[(d) Alcoholic cirrhosis (Liver rarely enlarged and (never excessively), indurated; venous plexus; splenic tumor very large, œdema; considerable ascites which by superadded (tuberculous) exudation may become strongly albuminous; subicterus; no pain; generally no bile pigment in the urine; functional insufficiency may be absent; gastro-intestinal catarrh and hæmorrhages).]

[(e) Cirrhosis in Banti's disease (Enlarged spleen, ascites, icterus, leukopenia).]

[Also: Leukæmia, pseudoleukæmia, splenic anæmia (Liver moderately enlarged and indurated, sharply demarcated, smooth, rarely ascites and icterus). Infectious hypertrophic cirrhosis (measles, scarlet fever, malaria) (Liver firm, at first painful, usually splenic tumor, icterus, epistaxis, and ascites). Acute yellow atrophy of the liver (first stage), liver carcinoma, hepatic cysts, hepatic echinococcus (Fluctuating tumor between umbilicus and ensiform process, continuously growing, not painful, smooth; good general condition; puncture fluid characteristic).]

NOTE.—In rachitis and myxœdema, enlargement of the liver to a moderate extent and without functional disturbance is frequently met with.

59. CONTRACTION OF THE LIVER

Demonstration: physiological behavior, *see No. 58.*

Simulated by gaseous distention of the intestine and effusion into the abdominal cavity.

Atrophy of the liver as part manifestation of marasmus.

[Atrophic interstitial cirrhosis in alcohol poisoning and syphilis, acute yellow atrophy of the liver; see *Hepatic Tumor* and *Icterus*.]

60. ENLARGED SPLEEN

Demonstration: Inspection and percussion usually reveal nothing reliable. Palpation as above (*No. 57*); the flat hand should advance from below toward the costal arch in the left anterior axillary line, where the finger tips carefully penetrate on inspiration; catch hold deeply and now watch for the downward displacement of the antero-inferior splenic pole during the next following inspirations; sometimes the splenic pole can only be reached by executing short downward thrusts with the palpating hand, while the other hand makes counterpressure at the costal arch. Right lateral position facilitates the demonstration. Palpation from in front down around the costal arch yields less satisfactory results in little children.

In the newborn the normal spleen is sometimes palpable; otherwise only if enlarged or displaced.

Simulated by fecal accumulation, displacement of spleen (left pleural exudate, floating spleen in chlorosis), perisplenitis, floating kidney, renal tumors and genuine tumors of other organs of the abdominal cavity (Spleen freely movable with inspiration, splenic tumors retain shape of spleen and grow in diagonal direction through the abdomen), tenth rib displaced, ribs depressed.

A. PRIMARY SPLENOMEGALY.

1. *Leukæmia*, lymphatic, and myeloid chronic (Usually firm, indurated, notchy, often protruding beyond the median line, not painful).—Pseudoleukæmia and simple anæmia of younger children; pseudoleukæmia of infancy (enormously enlarged, reaching to the true pelvis, hard as stone, with sharp borders, freely movable, indolent); Banti's disease (later on also hepatic œdema, ascites, icterus, leukopenia).
- [2. Genuine tumors: Myxoma, lipoma, lymphadenoma, cysts; enlargement according to change of position, perisplenitis, abscess formation, traumatism.]

B. SECONDARY SPLENOMEGALY.

1. *Acute*, accompanying acute infectious diseases (Moderately large, often painful, soft).—Typhoid (generally in the beginning of the second week; still palpable a long time during convalescence), malaria (frequently persistent), scarlet fever, influenza, ulcerative endocarditis, cerebrospinal meningitis, miliary tuberculosis, septic pyæmia in older children, Weil's disease, erysipelas; rarely in croupous

B. SECONDARY SPLENOMEGALY.—Continued

pneumonia (sometimes post-critical), sepsis neonatorum, measles, diphtheria, botulism.

2. *Chronic*, accompanying chronic pathological conditions (partly with fatty and amyloid degeneration) (Harder, firmer, mostly indolent):

- (a) Constitutional conditions.—Rachitis, scrofulosis, myxœdema, status thymicus.
- (b) Conditions involving congestion in the portal or general system of circulation.—Cardiac, pulmonary, and various hepatic affections. *See Hepatic Tumor.*
- (c) Chronic infectious diseases.—Hereditary and acquired syphilis (indurated enlarged spleen in children under 3 months is almost always syphilitic), tuberculosis in various localities, chronic gastro-enteritis of non-specific nature.

61. ENLARGED LIVER AND SPLEEN

1. *Subordinated*: Spleen enlarged through engorgement consequent upon impeded portal circulation; cirrhosis.

2. *Coördinated*: Amyloid, fatty degeneration, especially in chronic infectious diseases (tuberculosis, syphilis), engorgement (*e.g.*, in pericarditis), rachitis, Banti's disease.

62. TUMOR-LIKE PROTRUSIONS IN THE UMBILICAL REGION**A. CONGENITAL.**

1. Hernia of umbilical cord (Partly surrounded by amnion, only partly and with difficulty replaceable, containing intestines, and also sometimes solid abdominal organs).
- [2. Persistence, protrusion, and inversion of the ductus omphalomesentericus (Red, secreting, cystic tumor, sometimes communicating exteriorly; contents: intestinal secretion, often feces).]
- [3. Persistence and protrusion of the urachus, with possible prolapse of the bladder (Evacuation of urine from the tumor, demonstration of communication with the bladder through injection of colored liquid).]
4. Cuticular umbilicus and persistent umbilical trunk (Cylindrical, rather large navel stump, covered by puffy, overlapping ridges of infantile skin).

B. ACQUIRED DURING THE FIRST FEW WEEKS OF LIFE.

1. Umbilical hernia (Covered by normal skin, containing portions of intestine or omentum, easily replaceable, eliciting

B. ACQUIRED DURING THE FIRST FEW WEEKS OF LIFE.—*Continued*
a gurgling noise).—Myxidiocy, cretinism, rachitis, and conditions accompanied by coughing and straining.

2. Fungus, granuloma (Usually only as large as a pea, often pedunculated; by the side of a secreting umbilical wound; character of a granulation tumor).

3. Adenoma (from misplaced cells of the vitelline duct) (Rather large, smooth, indurated, and somewhat rapidly growing tumor).

[4. Other genuine tumors of the umbilicus, myxoma, and sarcoma.]

C. ACQUIRED AT LATER PERIODS.—Periumbilical phlegmons in tubercular peritonitis and ulcerative peritonitis, intestinal tuberculosis (Vesicular bulging of the skin over the navel with semilunar, tense œdema of the skin; later, percolation of fluid and perforation).

NOTE.—Owing to deficient closing of the navel ring, umbilical hernias may be acquired in later life through lack of abdominal space or increased pressure of the abdominal muscles. *Abdominal Hernia*, see No. 17.

63. UMBILICAL HÆMORRHAGES IN THE NEWBORN

(and hæmorrhages in the newborn generally)

A. TRAUMATISM THROUGH OBSTETRICAL OPERATIONS, asphyxia (Mostly hæmorrhages into the skin—thorax, back—from skin injuries—of the injured umbilical wound—and into internal organs: liver, kidney and adnexa, lung).

B. DISORDERS OF THE CIRCULATION, traceable to:

1. Deficient expansion of lung and engorgement of vessels.

2. Changes of the hepatic parenchyma, with engorgement (in the umbilical region).

3. Congenital malformation of the heart and hypoplasia of the blood vessel walls.

4. Embolic processes.

C. BLOOD CHANGES (generally with diminished coagulability).

1. Sepsis neonatorum (especially the causative factors of hæmorrhagic diathesis) (Slight, repeated hæmorrhages from the umbilical wound into the external skin, the kidney, from mucous membranes—intestine, nose, ear; from serous membranes).

[2. Hæmophilia.]

3. Congenital syphilis (“Hæmorrhagica”).

D. Hæmorrhages from the umbilical vessels through faulty ligation, in conjunction with the causes enumerated under *B* and *C*.

64. CORYZA

(an inflammatory swelling of the nasal mucous membrane)

Simulated by spontaneous secretion of cerebrospinal fluid through the nose in hydrocephalus.

A. ACUTE (With serous or mucopurulent secretion).**1. In the Newborn:**

- (a) Simple rhinitis (Not before 3 days after birth; secretion mucous, at least in the beginning; palate and faucial mucous membrane generally involved).—Catching cold, infection through dirty bath water or from the mother.
- (b) Gonorrhœal rhinitis (1 to 2 days after birth; secretion profuse, creamy-purulent, sometimes bloody; distinct stenotic manifestations, skin irritations; microscopic examination!).—Gonorrhœal discharge in the mother; frequently associated with ophthalmic blenorrhœa.
- (c) Rhinitis in sepsis neonatorum.

2. In Older Children:

- (a) Simple catarrhal rhinitis, or coryza (Watery, transparent secretion, later yellowish, mucous, mostly bilateral; hyperæmia of upper lip and tip of the nose; secretion ceasing at night).—Catching cold, contact infection, grease infection, inhalation of dust and poison; special tendency of pampered and lymphatic children (adenoid vegetations!).
- (b) Diphtheritic rhinitis (Especially in nurslings and younger children; serobloody, caustic secretion; considerable stenosis; œdema and excoriation of the labial skin unilaterally; sometimes formation of membrane demonstrable. Glandular enlargement; grave general manifestations, often of a septic nature).
- (c) Rhinitis in acute infectious diseases (concomitant):
 - (a) Measles (In the beginning, prodromal with conjunctivitis; at first spotty hyperæmia, usually without epistaxis; at times pseudomembranous coats).
 - (β) Scarlet fever (Generally only in the course of the disease, with scarlatinal diphtheria of the nose and unfavorable termination).
 - (γ) German measles (Similarly to measles, but usually less intense).
 - (δ) Influenza (Acute onset; high fever; rapid transition into profuse ulceration and leading to bronchitic and otitic manifestations; bacteriological demonstration and factor of contagion!).
 - (ε) Whooping-cough (Prodromal).

A. ACUTE, etc.—Continued

- (c) Erysipelas, cerebrospinal meningitis, putrid infection, hay fever, croupous pneumonia (at the time of the crisis), [enanthema of varicella on the nasal mucosa (?), gonorrhœa].
- (d) Rhinitis from foreign bodies (Strictly unilateral, often bloody secretion, severe pain and nervous manifestations, rhinoscopical examination!); also insects and worms!
- (e) Toxic rhinitis.—Poisoning by bromide, iodide, arsenic.

B. CHRONIC, EXACERBATING AND RECURRING (Watery, mucous, or suppurative secretion, odorless, or with sweetish or fetid odor).

Predisposing causes: Dust-laden air, crooked septum, hypertrophy of the turbinated bones, adenoid vegetations.

- (a) Chronic rhinitis with hypertrophy of the mucosa (Odorless secretion, or with only sweetish odor; tenacious, dry, forming no large crusts; submucous œdema; erosions, scabs; stenosis, paroxysmal sneezing, coughing; rarely osseous necrosis).—After acute processes in constitutional affections (torpid scrofulosis, lymphatism, arthritism,—together with adenoid vegetations), and in affections of the accessory cavities.
- (b) Chronic rhinitis with atrophy of the mucosa, ozæna (Greenish, strongly fetid secretion, forming crusts, anosmia; no stenosis.—Syphilis, scrofulosis.
- (c) Syphilitic coryza (especially of nurslings) (At first, dry swelling with wheezing and snuffing; later, slightly bloody-serous secretion, swelling of mucosa and submucosa, stenosis, sometimes osseous necrosis; epistaxis and fissure formation; slow afebrile course; no bronchitic complication; frequently noticeable at birth, and in most cases during the first 4 weeks of life as first sign of the affection; later on, manifestations of infiltration at the upper lip, saddle-nose, “facies syphilitica”).
- (d) Syphilis of the nose (Ulceration, osseous necrosis).
- (e) Chronic croupous rhinitis (non-diphtheritic) (Prolonged non-contagious affection with glandular enlargement, running an almost afebrile course under the manifestations of an ordinary cold in the head; whitish yellow fibrinous deposits, without tendency to expansion, without Löffler bacillus *).

* Presumably there are also true diphtheritic processes with Löffler bacillus, following a similar course (abated infection).

B. CHRONIC, EXACERBATING AND RECURRING, etc.—Continued

- (f) Foreign bodies in the nose (Chronic, always unilateral, mostly fetid, ulcerous secretion).
- (g) Polypi (Long continued one-sided coryza, nasal twang, mouth breathing, lachrymation, smelling and hearing impaired, few local pains, headache; only after 4 years of age).

65. NASAL STENOSIS

Snuffing, breathing through the mouth, which is always kept open (with secondary malformations of the cranial and facial skeleton, and aprosexia; see under *Adenoid Vegetations, Pharyngeal Stenosis*). Inability to suck in nurslings, paroxysms of dyspnoea, cough, asthma, defective development of the thorax. (Tendency to affection of the respiratory mucous membranes.) "Nasal" twang in speaking, disorders of the smelling and hearing functions.

A. CONGENITAL.

- 1. Congenital obliteration or narrowness of the nasal passages (membranous or osseous, unilateral or bilateral) (Probing or air insufflation elicits resistance).
- [2. Adenoid vegetations (mucosa dry) and syphilitic rhinitis, sometimes congenital.]
- 3. Narrowness in myxœdema and chondrodystrophy (Short, flat, broad nasal bridge, nasal skeleton as if pressed into the face, no inflammatory manifestations, noise more like snoring than snuffing).

B. ACQUIRED.

- 1. Acquired crooked curvature, thickening and distention of the nasal septum (Probing and inspection!).
- 2. Hypostaphilia, adenoid vegetations.
- 3. Traumatism, hæmatoma accompanying traumatic or acute infections, abscess, syphilis, tuberculosis, lupus.
- 4. Nearly all acute and chronic inflammatory processes of the nasal mucous membrane (see *Disorders of the Nasal Membrane*).
- 5. Polypus (Never starting from the nasal septum, generally high position, mobile).
- 6. Foreign bodies (Sometimes latent for a long time, unilateral fetid pyorrhœa with blood striæ, trigeminus neuralgia).

66. SNEEZING

- 1. All nasal affections, especially sneezing from hay fever; measles and whooping-cough (like vicarious coughing?).
- 2. Irritation of strong light (in newborn).
- 3. Habitual in neuro-arthritis, asthma.

67. SEE TABLE FOLLOWING

68. STENOSIS OF THE LARYNX

Prolonged inspiration, inspiratory dyspnœa, retraction of the intercostal spaces, jugulum, and epigastrium, and considerable downward flexion of the larynx, also stridor in inspiration; the latter palpable as buzzing bruit. Expiration almost always normal; head retroflexed; play of the alæ and action of other auxiliary respiratory muscles. In most cases, voice hoarse.

Laryngoscopic examination in younger children technically very difficult, perhaps facilitated by the aid of local anæsthesia or under complete narcosis.

I. CONGENITAL, *see Congenital Stridor*

II. ACQUIRED

A. SUDDEN ONSET, duration at the most 1 to 2 hours.

Simulated by expiratory apnœa; occurring with laryngospasm in rachitis and in angry children (semi-voluntary, interspersed with furious crying).

1. *Pseudocroup*, or *spasmodic laryngitis* (Unexpected, sudden onset, almost always at night; voice entirely or nearly clear; cough rough, barking; larynx not painful; after the attack there is only coughing and snoring; often fever, usually coryza, no angina, no acute lymphadenitis; only in children between 1½, and 6 years; tendency to recurrence.—In adenoid vegetations, difficult dentition, hereditary tendency.
2. *Laryngospasm* (Sudden occurrence, often without any perceptible cause or after mental excitement; duration short; piping or crowing inspiration, sometimes cyanosis, cessation of respiration; voice and inspiration clear in the intervals; no cough; no fever; sometimes convulsions; constitutional signs, symptoms of tetany; occurrence almost exclusively during the first 2 years of life; relapses!).—Tetany, chronic hydrocephalus, and other organic cerebral affections; otherwise mostly joint action of a constitutional and an occasional factor; "spasmophilic" diathesis (in rachitis, disorders of nutrition, etc.), "tetanoid state"—local, laryngeal irritation through various pathological conditions, pressure in the jugulum, enlargement of the bronchial lymph-nodes.
3. *Foreign Bodies* (*see below*).

67. EPISTAXIS, BLEEDING OF THE NOSE

(In older children ¹)

Stimulated by bleeding from the mouth, fauces, etc. The quantity of the blood is frequently overestimated by the parents.

	Fluxionary.	Congestive.		
	Hyperemia of the nasal mucous membrane and rhexia.			Hemorrhagic diatheses and kindred conditions (with increased liability to tears of the walls of the vessels and diminished coagulability of the blood).
A. Accidental, occasional hemorrhage.	Prodromal in acute infections, especially typhoid, influenza, pneumonia, measles, cerebrospinal meningitis, erysipelas, polyarthritis, endocarditis, malaria, mumps, whooping-cough.	Sinus thrombosis (especially sinus longitudinalis).	Septic conditions in the course of many various infections, such as diphtheria, scarlatina, influenza, septicopycemia in appendicitis, etc., botulism.	Fall on the nose, blow; foreign body in the nose.
	In other acute pathological conditions, as in nephritis.	Acute thyroiditis.	Acute leukemia.	Simple acute rhinitis (rare); diphtheritic rhinitis; exanthema in measles.
B. Habitual, frequently recurring hemorrhage; tendency to hemorrhage.	In overheating (e.g., in school), mental and bodily overexertion, use of alcohol.	Occasional violent coughing and sneezing.		
	Diabetes insipidus, orthostatic albuminuria, chronic nephritis, plethoria.	Cardiac and pulmonary affections, with engorgement of the general circulation.	Hemophilia, morbus maculosus; chronic leukemia, pernicious anemia, pseudoleukemia and leukemia simplex, Hodgkin's disease, infantile scurvy, chlorosis.	Habitual picking at the nose—worms (?), chronic rhinitis (simple, syphilitic, serofluous), polypi. Lupus. Deformity of the septum.
	Sudden growth at puberty (?).	Struma, adenoid vegetations, whooping-cough, ² and other lingering cough affections, enlarged bronchial glands.	Cirrhosis and degeneration of the liver.	
	Viciousness for menstruation.			

¹ Bleeding of the nose in the newborn; causes same as umbilical bleeding (q.v.).² Here perhaps also injury of the wall of the vessel.

B. GENERALLY OCCURRING LESS SUDDENLY, lasting for days or weeks.

1. *Diphtheria* (possibly primary diphtheritic croup) (Gradual beginning, slow progression of the dyspnoea; generally preceded by nasal or pharyngeal catarrh, which may still exist; cough more hoarse than barking, aphonia, fever; sometimes expectoration of membranes with Löffler bacilli).

2. *Mucous and Submucous Laryngitis*, in glottis or subglottis (Leading more rapidly to dyspnoea than does diphtheria, but not so steadily progressive as the latter; voice and cough rough, not aphonic as in diphtheria; sensitiveness to pressure on larynx; no Löffler bacilli):

(a) Non-specific, acute or exacerbating, also cyclically recurring (Rough, barking cough, rather sudden, occurring in paroxysms; voice hoarse, seldom clear; long continued dyspnoea of an obstinate nature; considerable thoracic retraction, stridor, and feeble respiratory murmur; fever).—Especially in adenoid vegetations.

(b) Accompanying acute infectious diseases; not infrequently with termination in perichondritis of the larynx and tracheal abscess (Circumscribed pain on pressure, œdema, hectic fever, fluctuating tumor):

(a) In measles:

(i) Membranous: Diphtheria complicating measles (Generally occurring after the eruption).

(ii) Non-membranous, simply enanthematically catarrhal: Early croup in measles (prodromal; fever, barking cough, slight stenosis, agitation, Koplik's spots).

(iii) Non-membranous, with œdema and maculofibrinous affection of the mucous membrane, finally formation of ulcers; Croup in measles (Often grave stenosis, with unfavorable course and complications, usually together with maculofibrinous stomatitis).

(θ) In influenza (Sometimes in pseudocroup-like paroxysms; sometimes with spotted chorditis; exceptionally tough secretion, like liquid rubber; preceded by coryza).

[(γ) In varicella (first vesicles on the laryngeal mucosa, or only enanthema; also spasms) (Occurs early, before the exanthema of the skin, severe course; mostly fatal termination).]

B. GENERALLY OCCURRING LESS SUDDENLY, etc.—Continued

[(*d*) In and after typhoid (Perichondritis with abscess formation).]

(*e*) In whooping-cough (perhaps early symptom), erysipelas, malaria, [scarlet fever and mumps].

(*ζ*) Finally, proliferation of thrush fungus in the epiglottis and the false vocal cords (Aphonia, rarely distinct stenosis; thrush of the buccal cavity). [Descending stomatitis ulcerosa.]

3. *Œdema of the Glottis, or Œdema of the Arytæno-Epiglottic Folds* (Inspiratory stridor predominates, paroxysms of asphyxiation, cough often not rough, dry, painful, voice often changed very little or not at all, rarely aphonia, examination by palpation and speculum!);

(*a*) Laryngitis (accompanying acute infections, such as measles, influenza, whooping-cough, also anginas of non-specific nature, erysipelas).

(*b*) Grave ulcerative processes in the neighborhood.—Angina Ludovici, phlegmons, retropharyngeal and peritracheal abscess, etc.

(*c*) Tuberculosis and laryngeal syphilis.

(*d*) Urticaria.

(*e*) Serum disease (In most cases simultaneously with other manifestations of the serum disease; subsiding spontaneously in 1 to 2 days; simulates croup relapse in diphtheria).

(*f*) Drug Poisoning; *e.g.*, with iodide (iodoform), bromide.

(*g*) Scalding, corrosion (Buccal mucous membrane!).

(*h*) Renal and cardiac affections with general dropsy.

(*i*) On the ground of neuropathic disposition.

4. *Foreign Bodies* (Often sudden onset, momentarily grave paroxysm of asphyxiation, followed by moderate inspiratory and expiratory dyspnoea, rough respiration, stridor; spastic, barking cough often with bloody expectoration; at first afebrile; result of palpation!).

5. *Compression by Rapidly Growing "Tumors"; e.g.* retropharyngeal abscesses.

[6. *Hysteria.*]

C. STENOSIS, OCCURRING GRADUALLY, PERSISTING FOR MONTHS OR PERHAPS YEARS, PROGRESSIVE OR REMITTENT, FREQUENTLY RE-CURRING.

1. *Laryngeal Syphilis* (other specific manifestations!):

(*a*) Early form (Early and interstitial processes in nurslings of the first 3 months).

C. STENOSIS, etc.—*Continued*

- (b) Late form (Condylomata and gummata, especially of the glottis of older children; always over 2 years, generally over 10 years).
- 2. *Tumors, Papillomata, often Congenital, Generally on the True Vocal Cords* (Early hoarseness, cough, and attacks of asphyxiation, later progressive stenosis, stridor, chiefly inspiratory, audible especially at night; all manifestations may remit; sometimes smaller ulcers are also situated in the fauces):
 - (a) Granuloma after recurring laryngitis and operation for stenosis.
 - [(b) Fibroma, enchondroma, cysts.]
 - (c) Goitre, tumors of the lymphatic glands, abscesses of the neck.
- 3. *Disorders of Innervation: Paralysis of the Crico-Arytenoid Muscle.* Bilateral paralysis of the posticus (Inspiratory dyspnoea with stridor, expiration and normal phonation being unimpeded). Bilateral paralysis of the entire recurrent laryngeal nerve (Leads, in small children only, to moderate stenosis; at the same time total aphonia and inability to cough).—After typhoid, diphtheria, influenza, inflammations, foreign bodies, traumatism, poisonings, and cerebral affections; partial recurrent paralysis also in mediastinal conditions:
 - (a) Recurring spastic conditions as reflexes in nasal affections.
- [4. *Chronic Laryngitis* (rarely distinct stenosis).]
- [5. *Laryngeal Tuberculosis and Lupus* (only in grave cases of older children).]

69. LARYNGEAL STENOSIS WITH MEMBRANOUS COATING OF THE FAUCES

- 1. Diphtheria (suspicion also justified when measles is present); former diphtheria, pseudo-recurrence of croup and tonsillar coats; simulated by "serum disease" (serum exanthema and urticarial oedema of the glottis).
- [2. Scarlet fever (rarely involvement of the larynx).]
- 3. Pseudodiphtheria of the newborn.
- [4. Angina and pseudomembranous non-diphtheritic laryngitis (Distinguishable from diphtheria only by negative bacteriological findings and the further course).]
- [5. Maculofibrinous stomatitis and laryngitis in measles.]

70. APHONIA

Hoarseness, roughness until complete loss of the voice or the cough.

1. Acute and chronic inflammatory processes in the larynx.—Simple acute laryngitis (smoke, scalding, catarrh, spasmodic laryngitis) (Voice may remain clear, cough however being rough*). Diphtheria, laryngeal typhoid, sepsis neonatorum, laryngeal thrush. Simple chronic laryngitis, especially in adenoid vegetations (Voice either simply husky or rough, continuous or intermittent, worse in the morning and after efforts). Laryngitis of measles.

2. Syphilis and laryngeal tuberculosis; the former even in the youngest nurslings (Voice mostly dull, rough, bleating, or hoarse).

3. Paralysis (post-diphtheritic), hysteria, bulbar processes; paralysis of recurrent laryngeal nerve in mediastinal processes (Voice hoarse, husky; often intermittent aphonia).

4. Laryngeal tumors: Papillomata [and fibromata] (Voice husky, hoarse, or rough; manifestations intermittent).

5. Laryngeal œdema (*e.g.*, in whooping-cough) (Voice not always changed, sometimes rough, sometimes complete aphonia).

6. Exsiccation; cholera infantum; atrophy of nurslings.

7. Extreme bodily debility. Continuous crying.

71. TRACHEAL AND BRONCHIAL STENOSIS

Dyspnœa, almost purely inspiratory; respiration often diminished, laryngeal movement in respiration increased only slightly or not at all; inspiratory thoracic retractions.

Simulated by a stenosis in the higher portions of the air tracts; differentiation, *see under Dyspnœa*.

Tracheal stenosis: Loud stridor, often severe dyspnœa.

Bronchial stenosis: Generally less pronounced stridor and less severe dyspnœa; over one lung or certain parts of the same, weakened fremitus and respiration, no dulness of sound.

A. ACCIDENTAL (Displacement, obstruction of lumen).

1. Descending diphtheria (Always accompanying laryngeal diphtheria; to be assumed if intubation in diphtheritic laryngeal stenosis remains without effect and obstruction of the tube is excluded).

2. Tracheitis and bronchitis of non-specific nature with secretion; catarrhal and fibrino-inflammatory processes, also in the presence of infections. Bronchiolitis (of rachitic patients).

* Roughness of cough: Change of the mucous membrane at the laryngeal entrance and in the trachea; subglottic and supraglottic laryngitis; glottis clear. Roughness of voice: change of the glottis.

A. ACCIDENTAL, etc.—Continued

3. Foreign bodies in the trachea and the bronchi (or in the œsophagus) (Sudden onset with coughing or asphyxial paroxysms—the symptoms of the tracheal foreign body may exceptionally occur slowly; “flag noise”; in all cases Röntgen examination; usually inflammatory complications follow, with ulceration and gangrene).
4. Perforation by pus of suppurating glands.

B. HABITUAL (mostly compression).

- | | | |
|--|---|--|
| <ol style="list-style-type: none"> 1. Enlargement of bronchial lymph-nodes 2. Goitre and thyroiditis 3. Enlargement of the thymus | } | <p>May grow worse, asphyxial paroxysms threaten life. Characterization and differentiation, <i>see under Mediastinal Space Constriction.</i></p> |
|--|---|--|
4. Dilatation of heart and pericarditis.
 5. Cicatrization after tracheotomy, ulcers from cannula or tube.

72. CONGENITAL STRIDOR

Respiratory stridor existing from birth (often not noticed until several days old), possibly remitting; may be followed perhaps by thoracic deformity.

Simulated by the “grunting” of myxœdematous and mongoloid patients.

1. Thymogenous (especially in hereditary syphilis, through disease of the thymus, or in vicarious hypertrophy in splenic affections; status thymicus) (Stridor more inspiratory than expiratory, exacerbated in horizontal position, during sleep, and uneasiness; slight laryngeal movement, slight retraction in the hypochondrium, paroxysms of dyspnoea and cyanosis; deep tone in crying; characteristic dulness and shadow in radiograph (*see No. 79*); general condition affected; intubation generally useless; receding spontaneously at the end of the second year of life).

2. Strumogenous (congenital vascular goitre; endemic) (Often grave asphyxia at birth, manifestations of congestion with violent and possibly fatal dyspnoea; symptoms recede progressively, and disappear spontaneously within 2 or 3 weeks; œdema of the neck).

3. True congenital stridor (Almost exclusively inspiratory, croaking stridor with thoracic retractions in forced respiration, but without particularly grave dyspnoea and cyanosis; when at rest remittent, voice clear, with high sounding cock’s crow; general condition only slightly disturbed, epiglottis with considerable longitudinal curvature. Intubation with short tubes is of benefit. Spontaneous recession at the end of the second year of life).—Dysplasia of the superior laryngeal orifice, or abnormal relaxation of the walls, perhaps in relation to the disturbance of the coördinated muscular play in respiration.

[4. Lymphadenogenous (in exceptional cases, occurring in the first month of life) (Exclusively expiratory stridor, almost entirely ceasing during sleep and on inclining the head forward; deep tone of voice, dullness; general condition affected).]

[5. Pharyngeal (rarely congenital).—Adenoid vegetations.]

[6. Laryngeal (Rough voice, aphonia, dyspnoea, later on asphyxial paroxysms, inspiratory stridor).—Congenital laryngeal papillomata.

[7. Congenital (?) laryngeal syphilis.]

73. ANOMALIES OF RESPIRATION

Up to the fourth year of life abdominal respiration always physiological; later costo-abdominal; after the tenth year, in girls, thoracic respiration. During the first few months of life respiration often irregular, especially during sleep (pseudo-Cheyne-Stokes). Little children retract physiologically the point of insertion of the diaphragm.

Frequency of respiration: frequency of pulse=1:4 (less often 1:3 or 1:5).

1. Purely costal respiration.—Interference with the action of the diaphragm by pressure, pain, paralysis; pleuritis, peritonitis, organic neuroses (bulbar paralysis, post-diphtheritic neuritis), hysteria.

2. Purely abdominal respiration (in older children).—Emphysema, paralysis of the thoracic inspirators, rigidity of skin.

3. Asymmetric respiration.—Pneumonia, pleuritis, contraction of the lungs after chronic infiltration, unilateral bronchial stenosis, pulmonary tuberculosis (Dragging of the infraclavicular fossæ).

4. Superficial, retarded and irregular (especially simply intermittent) respiration (*see also Dyspnoea*).—Grave acute infections, cerebral processes (tumor, among others), disturbances of circulation in the brain (hydrocephaloid), "meningismus," meningitis; agony, coma, uræmia.

5. Superficial accelerated (and jerky) respiration.—Pleuritis, asphyxia, atelectasis of the newborn, bronchopneumonia, pulmonary apoplexy; hysteria, chlorosis.

6. Cheyne-Stokes type (of less importance than in adults).—The same causes as irregular respiration (*see above*); further, alkaloid intoxications; days and weeks prior to death in prematurely born weak children.

7. "Deep" ("acid") respiration.—Intoxications of ectogenous (acids) and endogenous origin (coma of diabetes, uræmia, grave acute intestinal conditions; autotoxic, cyclical vomiting with acetonuria); paralysis of the heart after diphtheria.

8. Dyspnoea, tachypnoea, and stenotic respiration, *see under Dyspnoea, No. 74*).

9. Inverse or jolting respiration (Pause after inspiration, accent on expiration).—Croupous and lobular pneumonia (especially in pleural involvement).

74. DYSPNŒA

Restricted, labored respiration in abnormally low, normally or abnormally high frequency of respiration (tachypnœa; the latter perhaps also by itself). Pause between expiration and inspiration always abridged. In the severer grades, forced erect position, auxiliary muscles of respiration in action, speech cut short, uneasiness, difficulty of breathing, anxiety, mydriasis, cyanosis, cold perspiration; pulse usually frequent, tense, arrhythmical, unequal (sometimes "intermittent inspiration"). Dilatation of the right heart. Œdema of the liver, oliguria, involuntary micturition.

Simulated by short breath in respiratory pains (certain pleural affections, fracture of the ribs, muscular rheumatism, trichinosis, peritonitis).

Rapid respiration normal during and after bodily exertion.

Occurrence general; absolute and relative insufficiency of gas exchange in the lungs.

I. CONGENITAL

Due to aspiration of amniotic fluid; severe traumatism during birth, pulmonary apoplexy, atelectasis neonatorum, congenital stridor (*q.v.*).

II. ACQUIRED

A. GENUINE DYSPNŒA: Pronounced restricted, labored, and prolonged inspiration or expiration with diminished frequency of respiration (frequency diminished because the impeded inspiration or expiration requires more time).

1. *Inspiratory Dyspnœa* (Impeded, prolonged inspiration; inspiratory retractions of the thorax, with normal pulmonary sound).

(a) Stenosis of the upper air tracts (Stridor, phenomena of aspiration of the parts situated below the stenosis (namely, episternal, epigastric, supraclavicular), retroflexion of the cerebral vertebral column).

(a) Nasal stenosis* (Dyspnœa usually only paroxysmal, picture lighter, since vicarious mouth-breathing intervenes). (*See No. 65*).

(β) Pharyngeal stenosis* (Snoring and fluttering respiratory murmur, which remains audible even with occlusion of the nose; dyspnœa usually constant, but paroxysmally aggravated; intercurrent spasm of the glottis; impediment visible and palpable). (*See No. 32*).

(γ) Laryngeal stenosis (Voice almost always hoarse, often complete aphonia, always inspiratory

* Usually only slight or medium grades of dyspnœa.

A. GENUINE DYSPNŒA: etc.—*Continued*

stridor, barking cough, very pronounced respiratory movement of the larynx, distinct acoustic character of the murmur, dyspnœa often of a high degree, expiration nearly always free, laryngoscopic examination!). (See No. 68).

(d) Tracheal and bronchial stenosis (Pronounced inspiratory dyspnœa, but also expiratory stridor, less marked inspiratory retractions, movement of the larynx in respiration wanting or slight, voice clear, head bent rather forward. Auscultation: piping bruit, non-respiration of certain parts of the lungs without change of pulmonary sound, fremitus diminished; sometimes extension of the lung on the healthy side; asymmetry of respiration; intubation usually without success). (See No. 71).

(b) Paralysis of Inspiratory Muscles.

(c) Thorax-rachitis, elevation of diaphragm through increased pressure (increased filling) of the abdomen. Meteorism, ascites.

2. *Expiratory Dyspnœa* (Impeded, prolonged expiration; no dulness over the lungs).

[(a) Pulmonary emphysema (not vicarious) (Physical signs of symmetric pulmonary distention, box sound, slight motion of the pulmonary borders, fremitus and vesicular respiration diminished, thoracic deformity!).]

[(b) Bronchial idiopathic asthma in neuro-arthritis and as a reflex in nasal, faucial, and bronchial affections (Piping respiratory murmur, otherwise no signs on auscultation, moist râles only in the latest stages; pulmonary distention, low position of diaphragm; pulse small, but tense; paroxysmal occurrence).]

(c) Spasm of the inspiratory muscles.—Diaphragmatic spasm in irritation of the phrenic nerve through exudative pericarditis; tetanus, tetany, epilepsy, hysteria, convulsions, Thomsen's disease.

[NOTE.—Very rarely, an expiratory dyspnœa occurs in stenosis of the upper air tracts, perhaps through displacement of membranes or tumors in front of the lower aperture of the glottis.]

B. MIXED DYSPNŒA (usually with tachypnœa) and conditions of simple tachypnœa.

1. *Various Affections of the Bronchioles, Lungs, and Costal*

B. MIXED DYSPNŒA, etc.—Continued

Pleura; namely: bronchiolitis, acute infiltrations (especially pneumonia after measles, whooping-cough, influenza), pulmonary œdema, acute miliary tuberculosis, exudative pleuritis, hydrothorax, pneumothorax (All with more or less characteristic physical signs; no stridor; in slowly developing compression of the lungs—pleurisy with effusion—dyspnœa often surprisingly slight; in miliary tuberculosis, few physical signs; cyanosis; (*dyspnœa et cyanosis sine materia*); in pulmonary œdema, gasping stertorous respiration).

2. *Cardiac Insufficiency and Lessened Pulmonary Mobility* "*Cardiac Asthma*": mitral insufficiency, congenital defects, degeneration of heart muscle, acute endocarditis, myocarditis, pericarditis (Often paroxysmal attacks with anxiousness and cyanosis; pulse small, soft, frequent; lessened pulmonary mobility, moderate distention; cardiac dulness increased.)
3. *Deficient Oxidation of the Blood* consequent upon diminished content of Hb.—All forms of anæmia, especially the pernicious; also, infantile scurvy, leukæmia (Blood examination! Dyspnœa almost solely on exertion.
4. *Irritation of the Respiratory Centre in the Medulla Oblongata*:
 - (a) Acute infections.
 - (b) Endogenous and ectogenous intoxications, especially acid poisoning.—Mineral acids; acid auto-intoxication; acetonæmia, diabetic coma; also, uræmia. Gas poisoning.
 - (c) Organic affections of the pons and medulla.—Hæmorrhages, tumors.
 - (d) Elevated blood temperature in fever and in heat stroke.
5. *Neuroses*.—Hysterical asthma; neurasthenia (especially if there is at the same time any (slight) organic lesion).

75. COUGH**I. WITHOUT ANY CHANGES OF THE PARENCHYMA OF THE LUNGS AND THE PLEURA**

A. WITHOUT (SEVERE) DYSPNŒA (At first dry, later usually becoming "loose," then expectoration of mucous, suppurative, or fibrinous masses (*Expectoration, see Nos. 77 and 78*); concomitant inflammatory catarrhal manifestations, starting from the mucosa of the upper air tracts; in most cases accidental occurrence).

1. *Nasal and Faucial Cough* (Short, light, dull cough, more frequently at night and in the morning; often repeated

A. WITHOUT (SEVERE) DYSPNŒA, etc.—Continued

every few seconds; faucial wall hyperæmic, granulated, no râles, sometimes vomiting and nausea; cough will not occur through pressure on the trachea; occurs along with hawking, also with — and instead of (?) — sneezing).— Chronic rhinitis, hay-fever cough; nasal papilloma; sub-acute and chronic pharyngitis of anæmic, scrofulous, and lymphatic children; hypertrophy of laryngeal and pharyngeal tonsils.

2. *Laryngeal Cough* (With a hoarse, barking sound and laryngeal pain).—Simple laryngitis (catching cold, dust, vapor), spasmodic laryngitis (*see Stenosis of the Larynx*, No. 68), laryngitis in infectious diseases (influenza, measles, *see next paragraph*), other laryngeal affections (foreign bodies, swallowing the wrong way, scalding, etc.).

3. *Tracheobronchial Cough* (Loud, painless, at first dry, tenacious mucus, râles usually audible, scratching feeling in the trachea):

(a) Acute catarrhal tracheobronchitis.

(α) Idiopathic.—Inhalation of dust, catching cold.

(β) In acute infections: Influenza (Cough very severe and dry, sometimes quite spastic, slowly “becoming loose”; high fever, conjunctivitis, coryza preceding; earache, nervous manifestations, vertigo, weakness, feeling ill, agrypnia, anorexia, source of contagion!). Measles (Spotted hyperæmia of the palatal mucous membrane, Koplik’s spots, catarrh of eyes and nose, sneezing, characteristic temperature curve). Whooping-cough (first stage) (Cough extremely obstinate and frequent, possibly with singultus and mucous vomiting, uninfluenced by opiates, occurring especially during the first hours of night, continuing the even tenor of its way for days and weeks; at the same time coryza, hyperæmia of the conjunctiva, leucocytosis, high specific gravity of urine). Descending diphtheria (croupous bronchitis). Typhoid (Dry, and rather extended diffuse bronchitis). Miliary tuberculosis.

(γ) Intoxications (iodine, bromide, etc.).

(δ) Serum disease.

(ε) Engorgement in cardiac insufficiency.

SUPPLEMENTARY. — [Fibrinous bronchitis, pseudo-membranous (Very violent cough with dyspnœa;

A. WITHOUT (SEVERE) DYSPNŒA, etc.—*Continued*

severe pains with fever, expectoration of branching fibrinous masses—bronchial exudate—later mucopurulent, blood-tinged sputum; in places over the lungs, soft respiration and dull sound, râles; often emaciation and perspiration, no Löffler bacilli).]

- (b) Subacute, relapsing and chronic tracheobronchitis (Intermittent, often violent cough, usually with thick, yellowish, purulent expectoration; occurring especially in the winter months in the morning, not at night; stertor; often numerous humming and piping râles, which are audible some distance away, and occur chiefly at the base, less frequently at the apices; general condition not bad, usually no fever, no pulmonary foci, long duration, small reaction, no tubercle bacilli).—Lymphatic diathesis, scrofulosis, anæmia, rachitis, insufficiency of cardiac valves.

- (c) Bronchiectasis (Cough loose, moist; considerable quantities of mucopurulent expectoration, seldom streaked with blood, sometimes fetid, amounts up to $\frac{1}{2}$ litre in 24 hours, its evacuation simulates vomiting; snarling and moist râles, rough respiration, gurgling and pseudocavernous bruits in the middle or at the base of a lung, and unchangeable dulness; sometimes involvement of the pulmonary parenchyma; often flourishing appearance and good general condition, drumstick fingers).

B. ACCOMPANIED BY VIOLENT PRIMARY DYSPNŒA; the latter very prominent. Attacks paroxysmal. Usually on constitutional basis or as a sequel to chronic affections, therefore habitual recurrence. No pronounced objective signs in the mucous membranes of the upper air tracts.

1. and 2. *Bronchiolic Cough* (Mixed dyspnœa, frequency of respiration often enormously increased, no stridor, non-sonorous, finely vesicular, inspiratory and expiratory musical râles, bilateral, diffuse; thoracic retractions; high fever; occurs especially in little children under 3 years); and

Asthmatic Cough (Dyspnœa expiratory or mixed, diminished frequency of respiration, piping respiration audible at a distance; onset sudden while awake; cough appearing only at the end of the paroxysm, as compared with dyspnœa;

B. ACCOMPANIED BY VIOLENT PRIMARY DYSPNŒA; etc.—*Continued*
distention of lungs and rigidity of thorax, little or no fever, duration of attack a few hours to 2 days):

- (a) Bronchiolitis of rachitis, acute and recurring, complicated: by attacks of pseudocroup; fibrinous and croupous bronchiolitis.
- (b) Pulmonary tuberculosis with the type of bronchiolitis.
- [(c) Idiopathic bronchial asthma in neuro-arthritis, lymphatic-degenerative diathesis;] "reflex asthma" in affections of the nose, fauces, bronchi (nasal polypi, adenoid vegetations, bronchiectasis), helminthiasis.
- (d) Cardiac asthma in congenital (and acquired) heart disease.
- (e) Dyspeptic, uræmic, and toxic asthma. (*See Dyspnœa, No. 74.*)

C. ACCOMPANIED BY DYSPNŒA, the latter however secondary as a sequel to cough; often accompanied by retching and vomiting; usually no pronounced objective finding at the mucous membranes of the upper air tracts.

1. *Spastic Cough* (Exceedingly violent, paroxysmal attacks, frequently at night with cyanosis bringing all expiratory muscles into play).

- (a) Whooping-cough (second stage) (Staccato and reprise*), vomiting or retching up of mucous masses, real expectoration of the coughed up glassy secretion, no fever, negative pulmonary signs; in the intervals between the attacks good general condition; ulcer of frænum, "facies pertussea," leucocytosis, examination of urine, failure of medication, course, occurrence at night).
- (b) False whooping-cough, "pertussoid."
 - (a) Granular pharyngitis, enlarged tonsils (without reprise*) (Attacks less severe, which can only seldom be regarded as whooping-cough).
 - (β) Enlarged tracheobronchial lymph-nodes (Rough sound of the cough, usually no reprise, no vomiting, shorter attacks, often hoarse voice, sometimes asthma; often physical signs over the lungs, piping inspiratory bruit, shallow respiration, seldom dulness over the nodes, no expectoration of filamentous masses; physical and functional signs continue to exist between the

* Reprise does not here mean the repetition of the coughing impulses, but the intermediate noisy stenotic inspiration.

C. ACCOMPANIED BY DYSPNŒA, etc.—*Continued*

- attacks; usually remitting fever, perspiration, emaciation, very obstinate behavior, chronic course, no catarrhal prodromal state, no factor of contagion, usually no occurrence at night).
- (γ) Chronic Bronchitis (Attacks short, without reprise, cough especially in the morning, not at night).
 - (δ) Every bronchitic process which occurs in the months following whooping-cough (history!)
 - (ε) Measles and influenza prodromals.
 - (ζ) Laryngospasm along with bronchitis (in which the cough produces the spasm), also pseudo-croup.
 - (η) Foreign bodies in the larynx (Without reprise, course!).
 - (θ) Periodical night cough (Attacks of violent, dry, spasmodic cough recurring every night 2 to 3 hours after going to sleep, in children who otherwise appear healthy).
 - [(ι) Hysteria (Attacks only at daytime).]

SUPPLEMENTARY.—“Nervous cough” with “laryngeal crisis” (Dry, tormenting, with precordial sensations, sometimes spastic, with laryngeal stenosis, usually paroxysmal; no distinct objective signs).

- (a) Central vagus irritation (medullary conditions) or peripheral (especially affections of the heart and pericardium).
- (b) Cardiac neuroses, hysteria; by way of “reflex” in intestinal parasites (? !).
- (c) Paralysis of recurrent laryngeal nerve (pericarditis, tuberculosis of bronchial nodes) may lead to hoarse, croaking, powerless coughing; open glottis!

II. IN AFFECTIONS OF THE LUNGS AND PLEURA

(See also *Pulmonary Infiltration*, Nos. 80 and 81)

1. Short, dry, painful, but long continued.—Incipient croupous pneumonia (also latent central pneumonia without physical signs) (along with rapid respiration, continuous high temperature, abdominal or pleural pains), pleuritis (often also—especially in the course of the disease—paroxysmal), pulmonary tuberculosis (tormenting cough, occurring especially in the morning, tenacious secretion), and others.

2. Moist, abundant.—Lobular, catarrhal, and hypostatic pneumonia; œdema, gangrene, pulmonary abscess, pulmonary tuberculosis (caseous pneumonia, phthisis), chronic pneumonia.

76. COUGH WITH VOMITING

1. Simulated by bronchiectasis.
2. Whooping-cough, influenza.
3. Chronic rhinitis, pharyngitis, and hypertrophy of the tonsils.

77. SPUTUM EXPECTORATION

Younger children certainly "expectorate," but do not eject the matter, as they swallow it as soon as it is brought up to the fauces or mouth. Children under 5 years therefore almost never produce expectoration spontaneously,—except perhaps in whooping-cough; also, older children expectorate rarely (pulmonary gangrene, pulmonary tuberculosis, bronchiectasis).

Technic of obtaining sputum: (a) After an attack of coughing, produced either spontaneously, or artificially by pharyngeal irritation, pressure on the jugulum, etc., wipe out the fauces with the aid of a sterile cotton-tipped applicator; (b) In case of need, introduce either a Nélaton catheter into the larynx, or intubate, taking the sputum from the eye or lumen of the tube; (c) Wash out the child's stomach before breakfast. [Passing a catheter or stomach tube down the œsophagus, then pinching it and driving it out will usually obtain sputum in the eye of the tube. Another method is to fix a piece of dry gauze in an artery clamp, then to excite a cough by touching the epiglottis with the gauze—The cough will usually throw some sputum up into the gauze.—La F.]

1. *Muco-glassy, Frothy, Grayish White, Tough.*—Acute tracheobronchitis, whooping-cough, influenza.
2. *Mucopurulent, Often Malodorous, but not Distinctly Fetid.*—Suppurative bronchitis, bronchiectasis (in large masses periodically removed), lung cavities in tuberculosis (often yellowish green to grass-green).
3. *Pure Pus, often Rancid and Fetid Smell.*—Perforated empyema, abscess of the lung, cavities; third stage of whooping-cough.
4. *Fetid-putrid.*—Pulmonary gangrene; perforated, communicating empyema, bronchiectasis.
5. *Seropurulent.*—Grayish green, three layers, discolored, of cadaverous smell).—Pulmonary gangrene.
6. *Serous*, usually muco-sanguineous.—Pulmonary cedema.
7. *Bloody* (simulation by blood from nose and mouth in epistaxis, stomatitis, etc.).

(a) Traces of blood frequently in children from 1 to 6 [for instance, rusty sputum in pneumonia].

[(b) Rather large quantities of blood; "hæmoptysis."—Congenital and acquired cardiac insufficiency (congestion of the lungs), infarct of the lungs (apoplexia pulmonaris

7. *Bloody, etc.—Continued*

neonatorum), pulmonary gangrene, pulmonary tuberculosis, pulmonary œdema, chronic pneumonia, whooping-cough, and thoracic traumatism. Bronchiectasis, croupous bronchitis and bronchiolitis, hæmorrhagic diathesis. Foreign bodies in the upper air tract. Vicarious menstrual hæmorrhage; hysteria.]

78. SPECIAL CONSTITUENTS OF THE SPUTUM

1. Bile pigment.—Icterus.
2. Tracheal and bronchial exudates (tubular or solid).—Descending diphtheria [fibrinous non-diphtherial bronchitis], croupous pneumonia.
3. Shreds of pulmonary tissue and elastic fibres.—Abscess and gangrene of the lungs, bronchiectasis with secondary pulmonary processes, tuberculosis.
4. Pieces of cartilage.—Ulcerative perichondritis in the upper respiratory tract.
5. Spiral and central filaments (exudates from the finest bronchioles and alveolar passages).—Bronchial asthma, capillary, fibrinous bronchitis congestive bronchitis, croupous pneumonia, tuberculous phthisis, infarct of the lungs.
6. Thrush and fungous growths.
7. Cells in cardiac insufficiency.—Induration of the lungs in mitral insufficiency, myocarditis, pericarditis.
8. Crystals of hæmatoidin, stearic acid, cholesterin, leucin, and tyrosin.—Abscess of the lungs, gangrene of the lungs, fetid bronchitis, empyema.
9. Charcot-Leyden crystals.—Bronchial asthma, bronchitis, tuberculosis, gangrene.

79. SPACE CONSTRICTION IN THE UPPER MEDIASTINUM

SYMPTOMS.—*First series:*—Manifestations dependent upon the presence of constricting factor ("tumor") itself: Dulness, shadows in the radiogram, bronchial respiration over the manubrium sterni.

Second series:—Manifestations dependent upon compression of neighboring organs:

- (a) Trachea, bronchi, lung: Inspiratory dyspnœa predominating, with diminished frequency of respiration, tracheal râles, and stridor; sometimes attacks of suffocation, pseudocroup-like (*see Dyspnœa, No. 74*); sharp bronchial or attenuated breathing, with or without slightly diminished note and lessened fremitus over various parts of the lungs (bronchi compressed either wholly or partly),

SYMPTOMS OF CONSTRICTION IN UPPER MEDIASTINUM.—Continued

- ægophony; hoarse, rough voice, aphonia; thoracic asymmetry in consequence of atrophy of poorly aerated lung.
- (b) Vessels (vena cava superior and innominate) and heart: Cyanosis of the face; collateral venous plexus at the upper anterior thoracic wall; œdema of the face, either unilateral or bilateral, also of the neck and arms; tinnitus aurium, vertigo; displacement of heart; Smith's phenomenon (venous murmur in extremely retroflexed neck).
- (c) Nerves (pneumogastric; recurrent laryngeal, pulmonary plexus, sympathetic): Paralysis of vocal cords with paroxysm of suffocation, spastic cough; dysphagia, vomiting, cardiac palpitation, tachycardia. Physiological dullness of thymus in children during the first year of life (see Fig. 2).

Simulated by pulmonary atelectasis, pericarditis, and pleurisy with effusion.

1. Thymus (Usually distinct dullness at typical area; anterior mediastinum most affected, compression of the trachea in front, compression of the other organs behind. Swollen follicles of the tongue, enlarged spleen, good state of nutrition; mostly younger children without fever).—Simple hypertrophy, or backward involution of the thymus, status thymicus.

[Inflammations, hæmorrhages, syphilis, tuberculosis, lymphoma, sarcoma, acromegaly.]

2. Lymphadenoma (Rarely pronounced dullness at the left sterno-clavicular articulation or behind and higher up intrascapularly; the posterior mediastinum mostly affected; nerves and vessels distinctly compressed. Often scrofulo-tuberculous constitution, cachexia, glandular tumors at neck and clavicle; predominating in older, frequently feverish children).—Tuberculous infiltration and caseation of the tracheo-bronchial and mediastinal lymph-nodes.

Simple inflammatory swellings, collaterally in inflammatory processes in the thoracic region (post-infectious pneumonia).

Leukæmia and pseudoleukæmia.

[Syphilis and genuine tumors.]

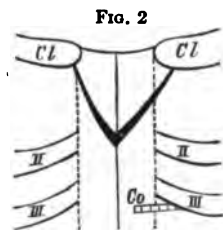


FIG. 2

Average thymus dullness from 22 cases aged 1 month to 1 year.

80. ACUTE INFILTRATION OF THE LUNGS

Especially: Tympanites or compact dullness, bronchial breathing, bronchophony, crepitation, abnormally strong transmission of the cardiac sounds, increased fremitus.

Demonstration: First inspect and palpate, then auscultate (with the free ear and with the binauricular stethoscope—the latter over places difficult to reach, like the point of the axilla, supraclavicular fossa; also in very young children and those difficult to move); finally, percussion. Determination of the pulmonary borders anteriorly and laterally in dorsal position, if possible; behind in the sitting posture. Percussion of infants in arms is not recommended; in any case, careful attention has to be paid to as symmetrical a position as possible. To determine the borders gentle and quick percussion: finger upon finger! Small pleximeters—the best suitably cut from india-rubber—are only to be recommended in older children. Percussion should be repeated over the same spot, *i.e.*, in various phases of respiration. Strong percussion is needed to demonstrate condition of deep seated tissues.

In auscultation and percussion the findings in symmetrical places on both halves of the thorax should be compared.

Physiological limitation of lungs and lobes in projection upon the thoracic wall.*

R.F., mammillary line: Sixth rib, upper border (more rarely lower border of the sixth or the fifth rib).

R.S. and L.S., median axillary line: Ninth rib, upper border (more rarely eighth intercostal space).

R.B. and L.B., by the side of the median line: Eleventh spinal process of the dorsal vertebræ (more rarely tenth or twelfth); inner scapular line: Tenth rib.

The lower borders of the lung run horizontal antero-posteriorly.

R.F., above the fourth rib, Upper lobe; below the fourth rib, Middle lobe.

L.F.: Upper lobe.

R.B. and L.B., above the spina scapulæ, Upper lobe; below the spina scapulæ, Lower lobe.

Healthy children under 8 years of age have in ordinary speaking no vocal fremitus (or, as compared to adults, only a minimal one). When they raise the voice or cry out, fremitus can be distinctly perceived with the ulnar border of the hand gently placed in position, or with the tips of the fingers.

I. INFILTRATION SIMULATED BY:

A. PHYSIOLOGICAL FINDINGS.

1. Supposed dulness R.B. below: Lower border of the healthy right lung behind higher than left; relative liver dulness! The musculature which lessens the sound and increases the resistance is in older children stronger right than left.

*R, right; L, left; F, front; S, side; B, behind.

A. PHYSIOLOGICAL FINDINGS.—*Continued*

2. Diminished sound in the right axillary line below: Frequently physiological, occasioned through stronger thoracic curvature.
3. Diminished sound from crying or from straining: Especially R.B. below, physiological.
4. "Rauchfuss dulness" by the side of the spinal cord, triangular, with the basis at the border of the liver, over healthy lung, if on the opposite side there is a pleural effusion of some magnitude. [Also known as Grocco's Triangle—La F.]
5. Bronchial breathing and bronchophony by the side of the spinal cord, especially at the right side of the upper dorsal vertebræ, physiological. Account should be taken of the physiological sharper "puerile" respiration, which to a physician who is accustomed only to the treatment of adult patients may simulate bronchial breathing. Bronchial breathing is transmitted from the affected side to the healthy side.

B. OTHER PATHOLOGICAL CONDITIONS.

1. Atelectases (Percussion varies; generally slightly diminished sound with lessened or missing murmur; inspiratory dyspnoea, finely vesicular, dry, crepitating, no high-pitched râles, bronchophony and bronchial breathing, increased fremitus; no fever, sometimes subnormal temperature; occurrence especially in nurslings and bed-ridden older children; in genuine atelectasis child is pale, cyanotic).—Debility, displacement of the (lower) air passages (rhinitis, capillary bronchitis, almost always in catarrhal pneumonia); mechanical respiratory insufficiency (rachitic thorax, meteorism, etc.).
2. Pulmonary œdema (Resonance rarely much reduced, usually only tympanitic, diminished resonance and bronchial breathing; very moist, high pitched râles bilaterally behind and below; frothy serobloody expectoration, dyspnoea, cyanosis, paroxysms of suffocation; pulse and heart!).
3. Pleural effusion (*see No. 82*).
4. Bronchial and mediastinal glandular tumors (*see Space Constriction in the Upper Mediastinum, No. 79*).
5. Hyperæmia of the lungs (influenza), congestion (whooping-cough).
6. Thoracic deformities (rachitis, scoliosis), rachitic deformity of the scapula, etc.

II. INFILTRATION OCCURS IN:

1. *Croupous Pneumonia* (General symptoms setting in suddenly,—dyspnœa, vomiting, continued fever, hyperæmia of cheeks,—the physical signs of which do not follow until from 2 to 6 days later; tympanitic dulness; infiltration demarcated by lobar regions, generally unilateral; at first little coughing; herpes, patellar reflex often absent; urinary examination characteristic; especially acetonuria. Cyclic course, crisis).—After catching cold, or injury (?):

- (a) Differences from the course in adults: Rarely chills and fever, pleural pain and crepitation, almost never sputum rusty (*see also differentiation from pleuritis, No. 82*), more frequently purely central infiltration.

[2. *Splenic Pneumonia* (*Grancher*), *Pseudopleuritis* (General manifestation as in No. 1); localization mostly at the left below, respiration not increased, fremitus disappears, compact dulness, sharp bronchial breathing, crepitation, ægophony, disappearance of cardiac impulse, Traube's space free.]

3. *Catarrhal and Hypostatic Pneumonia* (No sudden onset, no alarming initial manifestations, fever lower and more remittent; infiltration—often with emphysema—rising symmetrically from the base, less compact dulness, distinct bronchial breathing, many râles, short, frequent, and painful coughing, retractions, dyspnœa and cyanosis).—Rachitis, scrofulosis, descending bronchitis, aspiration, foreign bodies, cachexia.

4. *Pneumonia after Infectious Diseases:*

- (a) After measles (Often picture of croupous pneumonia, but without crisis, with soft small pulse and rapidly fatal or long dragging course; sometimes lobular, symmetrical foci, severe dyspnœa).
- (b) After influenza (Often picture of croupous pneumonia,—apical!—but always after the prodromes of influenza, later on spreading to other parts; at other times lobular type, “bronchoplegia,” croup, viscous sputum, prolonged course).
- (c) After diphtheria (Beginning with faucial involvement or primary croup).
- (d) In whooping-cough (Onset may simulate croupous pneumonia, more frequently lobular after formation of atelectasis with slowly progressing infiltration; prolonged, asthenic course; the coughing attacks disappear; loss of strength, dyspnœa, cyanosis).
- (e) In typhoid (Beginning sometimes like croupous pneumonia, with dyspnœa and grave general picture; usually the upper lobe infiltrated, further course lobular, also septic-metastatic).

4. Pneumonia after Infectious Diseases :—*Continued*

(f) In sepsis (especially of the newborn) (General manifestations similar to croupous pneumonia, but with disseminated foci; interstitial infiltration).

[(g) In and after scarlet fever, rheumatism, varicella, dysentery, etc.]

5. *Caseous (Tuberculous) Pneumonia, Acute* (Disproportionately severe general condition, prostration, tachycardia, dyspnoea, emaciation, vomiting, anorexia, diarrhoea, oedema, diazo reaction, continuous or intermittent progress; other pathological foci in the organism—intestine, peritoneum, meninges, skin—diagnosis according to age):

(a) Under the picture of catarrhal lobular pneumonia (the most frequent form!) (Uncertain prodromal symptoms for a long time; later on appearance of cavity symptoms).

(b) Under the picture of croupous pneumonia (But without crisis and resolution, with irregular, steep variations of temperature).

6. *Embolic Infarct of the Lungs, Apoplexy of the Lungs* (Sudden appearance and primary affection! Usually chills, hæmoptysis, without fever, painful frequent cough, dyspnoea, pleural friction; later, manifestations of infiltration).—Cardiac insufficiency (especially simple dilatation of the heart after infections!). Marasmus; tuberculosis, suppuration, burns.

7. *Acute Abscess of the Lungs* (Intermittent fever with chills; ulcerous sputum with tissue particles and crystals; cavity symptoms).—(Influenza) pneumonia, foreign bodies, involvement of neighboring organs, cardiac insufficiency.

81. SUBACUTE AND CHRONIC INFILTRATION OF THE LUNGS

Dulness; indistinct and weakened respiration, bronchial breathing, prolonged rough expiration, bronchophony, high-pitched râles, shrinking of the lung with thoracic deformity; displacement of the heart.

Simulated by pulmonary tumors and pure bronchiectasis.

1. *Chronic Pneumonia* (Sometimes "pseudotuberculous": hæmoptysis, debility, cavities, shrinking).—Termination of the acute forms, especially 1, 3, 4, a, b, and c.

2. *Chronic Tuberculosis:*

(a) Chronic pulmonary tuberculosis (Generally after the second dentition, at the hilus or apex, hectic fever, perspiration, diazo reaction, enlargement of bronchial lymph-nodes; see also No. 86).

(b) Chronic caseous pneumonia (Frequently in younger children).

[(c) Subacute and chronic miliary tuberculosis of the lungs seldom exhibits distinct manifestations of infiltration.]

3. *Chronic Abscess of the Lung and Gangrene of the Lung* (Severe general condition; clay-colored, Hippocratic face; usually circumscribed, often multiple foci; local signs vary according to the original affection and the condition of the cavities, which often appear rapidly. The profuse sputum, which forms three layers of sediment, contains blood, necrotic parenchymatous shreds, and all kinds of characteristic, microscopic organic constituents, and has a cadaverous odor).—Acute (measles, scarlet fever, diphtheria, typhoid) and chronic (tuberculosis) debilitating infections: beside this predisposing cause a local pulmonary affection (various pneumonias, bronchial dilatation, pulmonary apoplexy); also septicopyæmic processes with pulmonary embolism, and aspiration of fetid masses and other foreign bodies.

82. PLEURAL EFFUSION

Simulated by (a) *Physiological Conditions* (see under *Infiltration*). In healthy children the right thorax is from 1 to $1\frac{1}{2}$ cm. broader than the left.

(b) *Infiltration*, see table on page 115.

(c) *By Other Affections*: Considerable pericardial effusion with compression of the lungs (Pseudopleuritic signs disappearing through forward inclination). [Peripleuritis, pneumothorax, splenic and hepatic tumors (Very mobile in respiration, hepatic dulness higher in front than behind), genuine tumors of the lung, the mediastinum, and the pleura.]

1. *Exudative Pleuritis, Exudation*:

(a) Serous and simple serofibrinous pleuritis (Usually acute, seldom insidious onset, remittent fever, pain and cough, seldom in children under 5 years).—Various infectious diseases (polyarthritis, pneumonia, measles, scarlet fever, syphilis), catching cold.

(b) Tuberculous serofibrinous pleuritis (insidious onset, usually together with pulmonary manifestations, exudate often sterile, sometimes containing tubercle bacilli, sediment shows lymphocytosis).

(c) Suppurative pleuritis, Empyema (Exudate purulent and containing pyogenic organisms, often rapidly increasing and profuse; subjective symptoms marked, grave general picture; fever—remittent or intermitting—may be absent in the further course; seldom in children over 12 years of age).

(a) Pneumococcus empyema (Most frequent form, with usually sudden onset; in older children relatively favorable, with little general reaction; in nurslings generally taking a very severe course; pus often encysted, creamy, yellowish green, with diplococcus

DIFFERENTIAL DIAGNOSIS BETWEEN PULMONARY INFILTRATION
AND PLEURAL EFFUSION

	Infiltration. ¹	Exudation.	Transudation.
Fremitus (in speaking loudly or crying).	Increased (unless the secretion blocks the bronchi; examine for cough).	Distinctly diminished	Distinctly diminished.
Dulness (localization, condition, form, and course).	Distribution varying; at first tympanitic, later not tympanitic, sometimes absolute; resistance slight, Traube's space ² normal.	Gradually rising dull border, posteriorly higher than anteriorly, unilateral, almost never distinctly and rapidly movable. Dulness intense, tympanitic only at the border, strong resistance, Traube's space may be dull (if affection is on left side).	Dulness as in exudation, but bilaterally symmetrical, distinctly and rapidly movable.
Respiratory murmur and voice.	At first undecided, later bronchial respiration, loud bronchophony, voice sounds not reduced.	Usually bronchial respiration (often very loud), bronchophony and ægophony, even amphoric-metallic murmurs. Sound of respiration and voice usually not at first diminished. ³	As in exudation.
Crepitation	Rather seldom	None	None.
Râles	Usually small vesicles, sonorous.	None	None.
Friction	None	May be present	None.
Displacement of the neighboring organs.	Seldom recognizable	Distinct	Distinct.
Forced position of the body.	Often pronounced	Often (at first on the healthy side, later) on the affected side.	Usually not pronounced, possibly orthopnea.
Circumference of thorax.	Circumference in affection of an entire lung or large portions often measurably enlarged on the affected side.	At first circumference of the affected side smaller, later considerably enlarged. Intercostal spaces sometimes obliterated, displacement of the sternum from the median line of the body.	Bilaterally enlarged.
Thoracic excursion	Affected side does not appreciably remain behind.	On the affected side, respiration remains distinctly behind.	Both sides remain behind.
Temperature	High fever, often intermittent, chills, and crisis.	Irregular fever, never crisis.	Little or no fever.
Pain	Usually slight	Considerable pain in the sides.	None.
Dyspnea and tachypnea.	Frequently present	In slowly developing effusions often absent for a surprisingly long time, in rapidly developing effusions very pronounced.	As in exudation.
Sputum, expectoration, puncture fluid.	Usually absent; no blood, or but little.	Absent: exudate (For distinction of both see <i>Effusion of Abdominal Cavity</i> , No. 56.)	Absent: transudate.

¹Exceptional conditions in splenic pneumonia, see No. 80, 2.²Traube's space is at the anterior thoracic wall, bordered by left costal arch, hepatic and splenic dulness, and region of pulmonary resonance. Corresponds to walled portions of stomach, and after meals often yields absolute dulness in the healthy.³Only in long continued presence of abundant exudate does the respiratory murmur become softer or inaudible.

1. *Exudative Pleuritis, Exudation*.—*Continued*

lanceolatus).—Especially after croupous pneumonia and osteomyelitis, and as a concomitant manifestation of polyserositis in nurslings (not diagnosed pneumonia?).

(β) Streptococcus empyema (Rarer form, often rapidly progressing, under a grave—typhoid—general picture; pus more fluid, gray, often containing long streptococcus chains).—Scarlet fever (also with nephritis), measles, diphtheria, erysipelas, polyarthritis, pyæmia.

(γ) Empyema, with findings of various kinds of bacteria, staphylococci, bacteria of the colon group, etc.—Typhoid, influenza, whooping-cough, sepsis; inflammation of the bronchial glands.

(δ) Empyema in tuberculous mixed infections (Generally insidious onset and course, leads to amyloid degeneration of the organs and to perforation of the pus into the bronchial tree and exteriorly).—Caries of the vertebræ, pulmonary tuberculosis.

(d) Putrid and gangrenous pleuritis (Pus fetid, putrid, with various organisms).—Communication of the pleural sac with the atmospheric air; ulceration and gangrene of œsophagus, lungs, ribs, and peritoneum, traumatism, etc. Usually with pneumothorax.

2. *Hydrothorax, Transudation*.—Nervous affections, (acquired) insufficiency of cardiac valves, various kinds of cachexia.

83. DIMINISHED RESPIRATORY MURMUR

Respiration on the left side is physiologically weaker than on the right side (the left principal bronchus being narrower than the right one).

1. General bodily debility, paralysis of the inspiratory muscles, superficial respiration when there is pain and cough irritation.

2. Stenosis of the upper air tracts (also nose and pharynx). See Nos. 32, 65, 68 and 71.

3. Affections of the pulmonary and costal pleura, pulmonary atelectasis, thickened pleura, pulmonary tumors, cardiac hypertrophy (attenuation at the upper left).

84. "CRACKED POT" PERCUSSION SOUND

("Clattering coins" on thoracic percussion)

In strong percussion during expiration under physiological conditions.

In the most various pathological conditions; therefore without any particular semiotic value.

85. CAVITY PHENOMENA AND OTHER RARE SIGNS OF PERCUSSION AND AUSCULTATION

Diagnostic importance analogous to that in adults.

86. GENERAL INDICATIONS IN THE DIAGNOSIS OF TUBERCULOSIS

A. CONSTITUTIONAL FACTORS: Signs of scrofulosis, habitus phthisicus (thorax long, flat, narrow; hyposternal angle acute, ribs and intercostal spaces antero-inferiorly broad, arched like an arcade; shoulders bent forward, extremities and neck long and thin; tips of fingers puffed up), general chronic swelling of the lymph-glands, especially at the neck and the clavicle, in younger children often also in the shape of micro-polyadenia (of value only if the glands are typically arranged and are very hard and very small), dryness of the skin, exaggerated hairiness, especially between the shoulder blades, long eye-lashes, circular caries of the neck of the incisor teeth.

B. PREDISPOSING FACTORS: Opportunity for contagion, living together with coughing patients; slow recovery after measles and whooping-cough.

C. HEREDITARY FACTORS: Disease and death in members of the family (especially when syphilis is excluded), many dying of unknown diseases in the earliest years.

D. LONG PRODROMAL SIGNS OF THE AFFECTION: Protracted bronchitis, arthritis, chronic disorders of nutrition, anæmia; swelling of bronchial glands (*see No. 79*); variations of pulse and temperature. Other uncertain prodromal signs.

E. CONSECUTIVE GENERAL MANIFESTATIONS: General debility, disproportionately rapid decline of strength and considerable emaciation, hectic fever, long-continued fever of unknown origin, diazo reaction of the urine, chronic enlargement of spleen and liver.

F. FORMER SPECIFIC LOCAL AFFECTIONS: Articular and osseous affections, cicatrices, tuberculous nodules of the skin.

G. SPECIFIC REACTION TO TUBERCULIN* (and subcutaneous injection of normal salt solution) [Agglutinin for tubercle bacilli in the blood serum].

*** TECHNIC OF THE TUBERCULIN TEST:** An injection of 0.2 mg. of fresh Old-Tuberculin in aqueous solution under the skin of the arm in the case of a baby who is continuously free from fever; in the case of an older child up to 1 mg. is used. In the case of negative or dubious results, double and treble the dose is injected after a few days. In this way it is possible to increase the dose up to the maximum of 10 mg.

POSITIVE RESULT: 1. *General reaction.* Sharp rise in fever in from 4 to 12 (to 24) hours after injection. Difference between climax and normal temperature taken at the same time of day, other things being equal, = 1.8° F. (1°C.).

2. *Local Reaction:* Pronounced stimulation of the local pathological conditions; in fact, increase in the general symptoms (cough, expectoration, pain, exudations in the pleural cavity and abdomen, etc.).

3. *Puncture Reaction:* Inflammatory manifestations of the skin around the puncture wound, from slight redness to cedematous swelling of the entire arm with erysipelatous discolorations and great sensitiveness.

Each of these three forms of reaction is "specific" in itself; and when they appear distinctly, are almost absolute proof for the existence of a tuberculous pathological focus in the body.

H. DEMONSTRATION OF THE CAUSATIVE FACTOR:**1. Bacteriological:**

- (a) Sputum (to obtain same, *see No. 77*). Simple streak preparation; or, in the case of a negative finding, collect larger quantities, dissolve in a little hot brine, and centrifuge after addition of an equal quantity of alcohol. Pour off the fluid and wash the sediment twice with alcohol by shaking, sedimentation, and decanting. Stain with freshly prepared fuchsin-aniline water.
- (b) Urine: Streak preparation of the sediment produced with the addition of alcohol. Attentive search for smegma bacilli. (Catheter).
- (c) Exudates (pleura and peritoneum) should be allowed to coagulate, after which the coagulum is removed with the platinum loop, is dissolved in a little warm brine, and the mass is ready for treatment as described under (a).
- (d) Lumbar Puncture: The fluid is placed aside for coagulation. As soon as a coagulum has formed, it is carefully caught on a glass slide placed underneath, upon which it is fixed and stained. (By removing the mass with a platinum loop, a stringy substance is produced which can only with difficulty be distributed and cannot be stained uniformly.) The author did not obtain any distinct increase of the bacilli in the lumbar puncture fluid by exposing it to incubator temperature.

2. Animal Experiment: Guinea-pigs receive an intraperitoneal injection of 5-10 c.c. of the non-coagulated exudate.

NOTE.—Tubercle bacilli in the urine are no proof of urogenital tuberculosis, nor is their presence in the stomach and feces proof of gastro-intestinal tuberculosis!

87. PICTURE OF CATARRHAL INFLUENZA

Severe general affection, pronounced feeling of illness, pain in muscles and limbs, hyperæmia of fauces, multiple acute enlargement of the lymph-glands, violent cough which may be barking, often nasal catarrh; fever; also, possibly diarrhœa, various kinds of superficial erythema, epistaxis, enlarged spleen.

1. Measles (prodromal) [possibly scarlet fever or German measles] (Typical fever curve).

2. Atypical typhoid (Seldom coryza, usually relative bradycardia, leucopenia).

3. Incipient whooping-cough.

4. Incipient croupous pneumonia (Seldom nasal catarrh, usually sudden onset).
5. Miliary tuberculosis (No coryza).
6. Congestive bronchitis with chronic cardiac affection (Onset less sudden, without the irritating manifestations of the acute infection and without erythema).

88. ARRHYTHMIC PULSE, BRADYCARDIA, OR BOTH COMBINED

(including irregularity and dicrotic pulse)

Changes in the quality of the pulse are often difficult to interpret during the first year of life; later on they are of the same value as in the case of adults.

Physiological in younger children when asleep, in excitement (medical examination!), in sensations of cold, and in sensitive irritations. Pathological in general, due to various injuries to the cardiac nerve and muscle apparatus.

A. ORGANIC DEFECTS SITUATED IN THE HEART ITSELF.—Interstitial myocarditis in infectious diseases (sometimes no bradycardia!), mitral stenosis (especially in disorders of compensation). [Pure acute endocarditis and pericarditis only by way of exception.]

B. ORGANIC DEFECTS SITUATED IN THE INNERVATION APPARATUS.

1. *Cerebral Affections*, especially those involving cerebral pressure.—Tumor, chronic hydrocephalus, the majority of the varieties of meningitis, especially the second stage of the tuberculous kind (not the cerebrospinal), encephalitis and abscess, concussion of the brain (also in traumatisms acquired at birth), disturbances of circulation in the brain (conditions involving violent coughing, sinus thrombosis, hydrencephaloid, migraine).
2. *Spinal Affections*.—Acute anterior poliomyelitis I.
3. *Peripheral Affections*.—Vagus irritations through mediastinal processes; also left-sided pleurisy with effusion.

C. REFLEX EFFECT.—From various regions, especially originating from the abdominal area, particularly accompanied by severe pain; *e. g.*, colic, constipation, intestinal parasites, difficult dentition, dyspepsia, appendicitis.

D. GENERAL AFFECTIONS OF THE ORGANISM.

1. *Intoxications*.
 - (a) Ectogenous cardiac poisons: atropine, caffeine, digitalis, opium, CO, chloroform.
 - (b) Endogenous: intestinal acetonæmia, uræmia [cholæmia in icterus from biliary obstruction].
2. *Infections* (partly endotoxic, partly acting immediately on the myocardium), especially in the very beginning and

D. GENERAL AFFECTIONS OF THE ORGANISM.—Continued

during convalescence (pneumonia, typhoid, scarlet fever, diphtheria), but also in the course of the disease (polyarthritis, measles, typhoid, sepsis neonatorum).

3. *Constitutional and Primary Blood Diseases*.—Rachitis, anæmia, leukæmia, "arthritism," obesity.
4. *Collapse* (especially also after sepsis neonatorum with sclerœdema), asphyxia, loss of blood.
5. *Functional Neuroses*.—Chorea, epilepsy, eclampsia, hysteria, enuresis, masturbation.
6. *In Hunger*.—Pyloric stenosis, stricture of the œsophagus, atrophy.

[SUPPLEMENTARY: Idiopathic bradycardia and arrhythmic pulse in Stokes-Adams's disease (Attacks of vertigo, fainting spells, convulsions, sudden death).]

89. TACHYCARDIA

Causes in part only quantitatively different from those mentioned under No. 88.

A. ORGANIC DEFECTS SITUATED IN THE HEART ITSELF.—Acute inflammatory processes of the serous membranes of the heart and the myocardium, congenital and acquired defects (especially valvular insufficiency of the left ventricle), dilatation and weakness of the heart.

B. ORGANIC PARALYZING AFFECTIONS OF THE NERVE APPARATUS.—Cerebral affections, especially basal meningitis III, cerebrospinal meningitis in every stage, changes of substance and disturbances of circulation in the region of pons and medulla; peripheral vagus paralysis. (Mediastinum! Postinfectious neuritis.)

C. FEVER AS SUCH: (Elevation of body temperature by 1.8° F. (1° C.) in children is generally accompanied in the average of cases by an increase in the frequency of pulse by 15 to 20 beats a minute).

D. GENERAL AFFECTIONS IN THE ORGANISM.

1. Intoxications of an ectogenous and endogenous nature; for instance, toxæmia in ulcerations.
2. Infectious diseases.—Especially scarlet fever, diphtheria, miliary tuberculosis, pneumonia, polyarthritis, septico-pyæmia, tetanus, erysipelas, measles; seldom in typhoid.
3. Constitutional diseases.—Chlorosis, diabetes mellitus, "arthritism," affections of the thyroid gland.
4. After loss of blood (in hæmorrhagic diathesis).
5. Functional neuroses.—Exophthalmic goitre, hysteria, neurasthenia, epilepsy.

[Add to this: Idiopathic, essentially paroxysmal tachycardia (Excessively high frequency of pulse; embryocardia ap-

D. GENERAL AFFECTIONS OF THE ORGANISM.—*Continued*

pearing suddenly in complete health, in relapsing attacks of one-half to 14 days' duration).]

SUPPLEMENTARY: Pulsus paradoxus (Retardation and weakening of the pulse during inspiration.—Convalescence after infectious diseases, pericarditis, myocarditis and myodegeneration (heart sounds fainter during inspiration). Adherent pericardium. Intestinal autointoxication. Croup in diphtheria, pseudocroup. Vagus involvement in bulbar paralysis, mediastinal tumors.

90. VESSEL PHENOMENA

A. DILATATION OF VISIBLE VEINS OF THE SKIN.

1. *Congestion on Account of Impeded Blood Flow in Proximal Regions:*

(a) Dilatation of veins:

- (a) Of the neck and upper arm, especially those of the region of the jugular vein.—Mediastinal (tuberculous) glandular tumors.
- (β) Between the ear and the large fontanelle in the region of temples and nose.—Thrombosis of the longitudinal sinus.
- (γ) In the region of the nape of the neck.—Thrombosis of the sinus transversus.
- (δ) At the temples and the eyes.—Hydrocephalus, parotitis, thrombosis of the sinus cavernosus.
- (ε) Of the legs.—Thrombosis or compression of the femoral vein in chlorosis and cachexia.

2. *Vicarious, on Account of Impeded Flow in Collateral Regions:*

(a) Dilatation of veins of skin:

- (a) Of the cranium.—Hydrocephalus (especially syphilitic), serous meningitis, rachitic cranial deformity.
- (β) Of the anterior thoracic wall.—Mediastinal tumors. (Here at times even in quite healthy children.)
- (γ) Of the abdominal wall.—Affection of the liver, with congestion of the portal system, effusion into abdominal cavity.

(b) Dilatation of the skin veins of the entire body.—Acute leukæmia, hereditary syphilis (?), tuberculosis (?).

(c) (Obliteration of the external jugular vein; thrombosis of the sinus transversus.)

B. PULSATION IN THE VEINS.

1. Negative pulse of the neck veins, venous collapse in cardiac

B. PULSATION IN THE VEINS.—Continued

- systole, presystolic swelling.—Physiological; increased by general engorgement.
2. Positive pulse of the neck veins and hepatic veins (swelling in cardiac systole).—Insufficiency of the tricuspid valve; possibly also in myodegeneration and muscular insufficiency.
 3. Collapse of jugular veins in diastole of heart.—Exudative and adhesive pericarditis. Insufficiency of tricuspid valve, open foramen ovale.

C. VENOUS MURMURS.

1. *Of the Neck Veins* (Jugular veins, louder right than left; inspiratory louder than expiratory; continuous, but diastolic louder than systolic murmur; louder in compression, which can also be elicited by turning of the head to the other side!).
 - (a) Not very loud in the healthy (Generally compression is required to make it audible, and it is demonstrable only at the neck).
 - (b) Spontaneously loud (“Nonnensausen”—humming sound). Cachexia, anæmia, especially chlorosis; goitre (Audible down to the second or third rib and under the clavicles).
2. *Of Deeper Veins* (venæ anonymæ—and vena cava superior).
 - (a) Smith’s phenomenon (Venous murmur in the region of the manubrium sterni, appearing or greatly increased on bending the head backward).—Constriction of mediastinal space; usually enlargement of lymph-glands or of thymus.
 - (b) Spontaneous murmur (right—and left—of the sternum).—Cachexia, anæmia.

D. MURMURS OF THE NECK ARTERIES.—In older children (who, unlike the younger children, emit murmurs of the carotid and subclavian arteries) by compression with the stethoscope or by tumors; and murmurs transmitted from other arteries by valvular defects.

91. ANOMALIES OF THE CARDIAC IMPULSE

Demonstration: Inspection and palpation.

For physiological position, see Table on page 124.

A. EXTERIOR DISPLACEMENT.—Hypertrophy and dilatation of the right ventricle, contraction of the lung, exudative pleuritis and pneumothorax on the right; considerable filling of the abdomen with fluid, and intestinal convolutions distended by gas.

B. EXTERIOR AND INFERIOR DISPLACEMENT.—Hypertrophy and dilatation of the left ventricle. Pericardial effusion.

C. MEDIAL DISPLACEMENT (often with appearance of diffuse concussion in the region of the ensiform process).—Persistent embryonal position of the heart, transposed viscera. Thoracic deformity, contraction of lung and pleura, pleural effusion and pneumothorax on the left.

D. AMPLIFICATION AND REINFORCEMENT.—Palpitations (*see also Tachycardia, No. 89*).

1. Without any Marked Anomaly of Dulness and Murmurs:

(a) Physiological: In excitement, in bodily exertion.

(b) Pathological: Fever, congenital heart disease, acute and chronic endocarditis.

(c) At puberty in chlorosis and rapid growth.

(d) Neuroses and factors acting by way of reflex.—Hysteria, dyspepsia, exophthalmic goitre, neurasthenia, epilepsy, masturbation, helminthiasis, colic; paralysis of the vagus.

(e) Constitutional anomalies, obesity, diabetes.

(f) Intoxications, medicaments, and articles of diet.—Amyl nitrite, chloroform, alcohol, tea, coffee, etc.

2. With Abnormalities of Dulness (*see No. 92*).

E. EXPANSION AND DIMINUTION.—Acute endocarditis, exudative pericarditis.

F. DIMINUTION (sometimes in the healthy the cardiac impulse is very indistinct).—Exudative pericarditis (Reinforcement when bending forward), and adhesive. Myodegeneration, disturbance of compensation in heart disease; collapse, pleuritis on left side, emphysema, stenosis of aorta, dermal oedema at thoracic wall.

G. SYSTOLIC RETRACTION IN THE REGION OF THE CARDIAC IMPULSE (and in the epigastrium, with systolic wholesale bulging of the thoracic wall in the superior cardiac region, and possible diastolic impulse).—Rheumatic pericarditis. (Similar behavior occasionally in cardiac hypertrophy.)

ANOMALIES OF (ABSOLUTE) CARDIAC DULNESS.

Demonstration: Determination of borders by exceedingly gentle percussion of finger on finger. To determine the borders, the peculiarities of resistance are more important than are those of murmurs: palpatory percussion.

For physiological behavior, see the following Table and illustrations.

Simulated by extreme inspiration and expiration (cardiac dulness less dependent on the respiration phase than in adults); through absence of air in the lung in the neighborhood of the heart (atelectasis, infiltration); through pleural layers and exudation; through thoracic deformity (rachitis, scoliosis).

ORDINARY POSITION OF CARDIAC IMPULSE AND BORDERS OF CARDIAC DULNESS IN HEALTHY CHILDREN.

Cardiac impulse maximum.	In the newborn (first week).		In the suckling (first year).		In infancy (second, third, and fourth year).		In childhood (fifth to twelfth year).		In puberty (thirteenth to fifteenth year).	
	1-2 cm. laterally from the mammillar line.		0-1 cm. laterally from the mammillar line.		Mammillar line or somewhat medially.		Mammillar line or somewhat medially.		Medially from the mammillary line.	
Toward circumference.....	1-2 cm. laterally from the mammillar line.		0-1 cm. laterally from the mammillar line.		Mammillar line or somewhat medially.		Mammillar line or somewhat medially.		Medially from the mammillary line.	
Upward.....	Fourth intercostal space.		Fourth intercostal space.		Fourth intercostal space.		Fifth intercostal space.		Fifth intercostal space.	
Upper border.....	Third rib (upper or lower border).		Third rib (upper or lower border).		Third intercostal space (upper or lower border).		Fourth rib (upper or lower border).		Fourth rib (upper or lower border).	
Outer border (always medially from cardiac impulse).	Between mammillar and parasternal lines.		Between mammillar and parasternal lines.		Between.		Nearer the latter.		Nearer the latter.	
Inner border.....	Left sternal border.		Left sternal border.		Left sternal border.		Left sternal border.		Left sternal border.	
Width about	2 cm.	3 cm.	4 cm.		5-5.5 cm.		5-5.5 cm.		5-5.5 cm.	
Upper border.....	Second rib.		Second intercostal space.		Third rib, upper border.		Third rib, upper border.		Third rib, upper border.	
Outer border (somewhat laterally from impulse).	2 cm. laterally from mammillar line.		1-2 cm. laterally from mammillar line.		Mammillar line or somewhat laterally.		Mammillar line or somewhat laterally.		Mammillar line or somewhat laterally.	
Right border	Right parasternal line.		Right parasternal line.		Right parasternal line or somewhat medially.		Right parasternal line or somewhat medially.		Right parasternal line or somewhat medially.	
Breadth about.....	6-9 cm.		8-12 cm.		9-14 cm.		9-14 cm.		9-14 cm.	

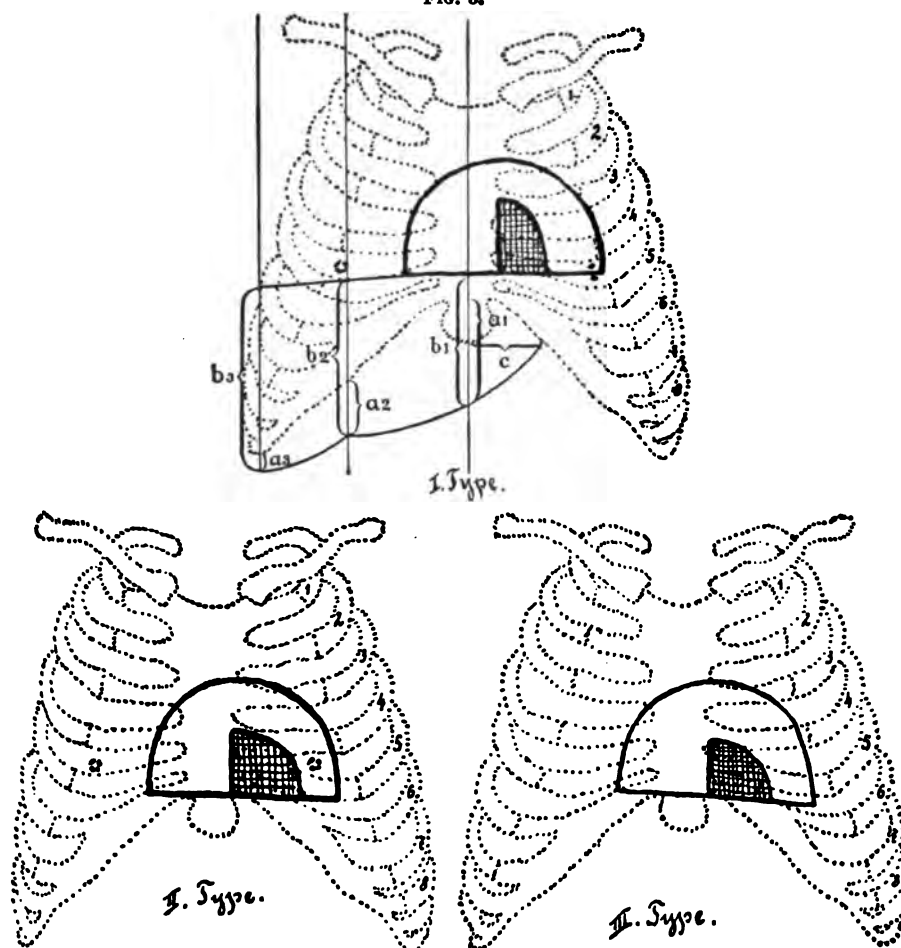
92. ENLARGEMENT OF (ABSOLUTE CARDIAC) DULNESS

(and increased resistance in the region of the relative dulness,
to which the absolute approaches)

Simulated by exaggerated percussion.

A. WITH STRONG CARDIAC IMPULSE.

FIG. 3.



Normal Areas of Heart Dulness
(after Hochsinger).

I. NEWBORN }
II. INFANCY } Shaded Area: Absolute heart dulness. Clear Area: Relative heart dulness.
III. CHILDHOOD }

1. *Hypertrophy of the Heart* (Increased tension of pulse, voursure; good general condition, fresh appearance):

(a) Of the left ventricle (Cardiac dulness enlarged downward and to the left; impulse displaced downward and to the left, broader, stronger, lifting; heart murmurs loud, especially of the II Aortic; strong pul-

A. WITH STRONG CARDIAC IMPULSE.—*Continued*

sation of the carotid and over the entire cardiac region; congestions, vertigo, eye-fluttering, tinnitus aurium).—Compensated valvular insufficiency. Endocarditis, pericarditis, chronic myocarditis, chronic nephritis.

(b) Of the right ventricle (Cardiac dulness expanded to the right,—and a little to the left,—impulse displaced, slightly to the left; heart murmurs loud, especially of the II pulmonic diffuse epigastric pulsation; tendency to bronchitis and dyspnoea).—Compensated valvular insufficiency, chronic endocarditis, pericarditis, and myocarditis; disturbance of the pulmonary circulation (infiltration, tuberculosis, bronchiectasis, whooping-cough, and cough diseases in general), rachitis.

(c) Of both ventricles.—Valvular defects and other cardiac affections; anæmia, chlorosis, scrofulosis, status thymicus; mental and corporal overexertion, precipitated development in the period of puberty, masturbation.

[2. *Acute Endocarditis in Fever* (Grave general condition, dyspnoea, cyanosis, arrhythmic pulse, bruits, expansion at first only to the left, later possibly also to the right).]

3. *Contraction of the Lung* (Other signs of pulmonary affection, borders immobile in respiration, strong visible pulsation of the denuded pulmonary artery).

4. *Thoracic Deformities*.

B. WITH WEAK CARDIAC IMPULSE:

1. *Dilatation of the Heart* (Sounds softer, especially the second vesicular sounds to disappearance, frequently relative murmurs of insufficiency; gallop rhythm and embryocardia; small soft pulse, dyspnoea, cyanosis, œdema).—Habitual: Usually after hypertrophy; same causes as this, or acting from the first under unfavorable circumstances (general debility, anæmia). Acute, accidental: Acute infectious diseases (ulcerous endocarditis, polyarthritides, diphtheria), acute nephritis.

(a) Distinction of dilatation of the right from the left heart is analogous to the differentiation in hypertrophy (see above, A. 1).

2. *Pericardial Effusion* (In larger effusions: enlargement of the cardiac dulness, especially upward to the second and third costal cartilage in triangular form, blunter hepaticpul-

B. WITH WEAK CARDIAC IMPULSE.—*Continued*

monary angle, diminution of the cardiac impulse, and its position within the dull area, manifestations of pulmonary compression; all these signs are particularly distinct in the recumbent position, less so in the sitting posture, and disappear on bending the body forward):

(a) Exudative pericarditis (Pain, sound of friction, fever; possibly characteristics of the puncture fluid, *see* No. 56).

(b) Hydropericardium (With hydrothorax and hydrops anasarca; characteristics of the puncture fluid, *see* No. 56).

3. *Adhesive Pericarditis* (Immobility and unchangeability of cardiac dullness and impulse by change of position—also in the radiogram; systolic retractions in the lower cardiac region, bulging in the upper; pulsus paradoxus, foetal rhythm, cardiodiastolic venous collapse).—After polyarthritis and in tuberculosis; (in the latter case few prominent cardiac symptoms; *see* *Hepatic Tumor*, No. 59).

4. *Acute Endocarditis* (*see* above).

SUPPLEMENTARY: 1. Narrowing of the (absolute) cardiac dullness.—Especially all states of distention of the lungs.

2. Change of position of the (absolute) cardiac dullness.—*See displacement of the cardiac impulse.*

3. Increased mobility of the (absolute) cardiac dullness through changing position of body. (Physiologically about 1 cm. through changing from one lateral position to the other).—Infectious myocarditis, in the period of convalescence after acute infections (diphtheria, typhoid); diminished blood pressure.

4. Diminished displaceability of the (absolute) cardiac dullness through changing position of body.—Exudative and adhesive pericarditis. Fixation through pleuritic cords and layers.

93. ANOMALIES OF THE CARDIAC SOUNDS

("prolongation," "roughness," and "impurity," of no semiotic importance)

Demonstration: Indirect auscultation. The best stethoscope for the pediatricist is the binauricular. Its advantages in pediatric practice, as compared with all other systems, are inestimable. It offers disadvantages only to the unpracticed physician. Small children sit on their mother's laps.

Infantile cardiac sounds are loud*) and are strongly transmitted to the back and abdomen! In the period of infancy the first murmur is accented also at the arterial ostia, and not only at the venous ones.

*Sequence of loudness (Hochsinger): I. Mi.; I. Tri.; I. Pu.; II. Pu.; II. Mi.; II. Tri.; I. Ao.; II. Ao.

1. *General Reinforcement of Murmurs:*

(a) Without hypertrophy.—Transitory in excitement, in cardiac neuroses, in the anæmia of puberty (palpitations); permanent in contraction of the lung, pulmonary infiltration (infiltrated parts conduct the sounds better), also anæmias.

(b) With hypertrophy (*see No. 92, A.*).

2. *Increased Strength of Single Murmurs:*

(a) First sound at the mitral.—Mitral stenosis.

(b) Second sound at the tricuspid and pulmonalis (in children, of very great importance).—Hypertrophy of the right ventricle with good cardiac power; overpressure in the small circulation; acute endocarditis, pulmonary affections, coughs.

(c) Second sound at the mitral and aortic.—Hypertrophy of the left ventricle, with good cardiac power.

(d) First and second sounds at the aortic and pulmonic.—Contraction of the lung.

3. *General Diminution of Murmurs* (can often be produced in the healthy by pressure with the stethoscope!).—Dilatation of the heart, cardiac debility (overexertion, hemorrhage; intoxication with CO₂, effect of other cardiac poisons; acute febrile infections, such as polyarthritis, typhoid; affections of the cardiac muscle), pulmonary emphysema, pericardial effusion, pleuritis, divers kinds of anæmia, incipient endocarditis.

4. *Diminution of Single Tones.*—Generally, on the occurrence of murmurs.

1st mitral sound	} Weakness of the heart and acute incipient endocarditis with and after infections (diphtheria, polyarthritis), dilatation, aortic (and mitral) insufficiency.
1st aortic sound	

2nd aortic sound.—Weakness of the heart, mitral and aortic stenosis.

2nd pulmonic sound	{ Pulmonary stenosis. (It should be observed that in healthy children the second pulmonic sound is not as loud as the first.)
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94. ABNORMAL RHYTHM OF THE HEART SOUNDS

1. Embryocardia, or foetal rhythm.—In the newborn physiological in young children often without importance. Otherwise, in commencing cardiac weakness, exudative and adhesive pericarditis, ulcerative endocarditis, dilatation of the heart, postinfectious myocarditis (diphtheria heart!), precursor of the galloping rhythm.

2. Simple sound splitting.—Frequently without importance and in healthy hearts; rare in nurslings; in the second pulmonic sound during violent crying. II mitral sound: mitral stenosis, diphtheria, typhoid and other infectious diseases, eccentric hypertrophy, [adhesive pericarditis].

3. True and pseudo-galloping rhythm (— — — and — — —).—Exceptionally, in healthy, greatly excited hearts; frequently simulated by the presence of pericardial murmurs along with dull sounds. Non-compensated heart disease, infectious myocarditis (especially in convalescence from diphtheria and typhoid), acute nephritis, acute ulcerous endocarditis, central or peripheral paralysis of the vagus.

95. SEE TABLE FOLLOWING

96. SYNDROME OF ACUTE ATTACK OF CARDIAC SYNCOPE

(myodegeneration, myocarditis, toxic lesion of the heart with acute dilatation)

Dulness enlarged*, cardiac impulse weak*, epigastric pulsation, sounds dull, split; first sound at the mitral almost inaudible, at all events murmurs of relative insufficiency (audible at first at the pulmonic then also at the apex); pulse small, arrhythmic, unequal*, bradycardia*, tachycardia; embryocardia, gallop rhythm; dyspnoea, beginning pulmonary oedema; repeated violent vomiting, abdominal pain, enlargement of the liver; oliguria, albuminuria; after jactitation, immobility, exhaustion, pallor, cyanosis, cold extremities, fainting.

Simulated by acute cardiac inflammations, especially of the serous membranes of the cardiac wall (*see No. 157*); further by typhoid perforation of the intestine, internal hæmorrhages, collapse in the bath, etc.

1. *Without Preceding Chronic Endocarditis* (II pulmonic sound not accented):

- (a) Acute infections.—Diphtheria (usually end of first to beginning of third week), scarlet fever and typhoid (usually between end of second and beginning of fourth week), polyarthritides, erysipelas, pneumonia, miliary tuberculosis, severe whooping-cough.
- (b) Intoxications and poisons.—Alcohol, lead, phosphorus, uræmia, cholæmia, burns of the skin.
- (c) Constitutional affections.—Grave anæmia, diabetes.
- (d) Chronic infections.—Hereditary syphilis and tuberculosis.
- (e) Exhausting effect of muscular work.—Chorea, severe whooping-cough.

2. *After Chronic Endocarditis* (II pulmonic sound accented).—Failure of compensation in valvular insufficiency.

3. *Exudative Pericarditis*, appearing rapidly.

97. SYNDROME OF CONTINUOUS MECHANICAL DISTURBANCE OF THE GENERAL CIRCULATION

("asystolia," "myasthenia cordis"—rare in infancy)

1. Small, soft, irregular, and frequent pulse.
2. Painful, hard, enlarged, pulsating liver (passively congested liver).

* Signs which are frequently observed as first signs.

95. CARDIAC

(Simulated by venous murmurs (*q.v.*, No. 90), and by respiratory sounds, if these

	Endocardial.		Pericardial.
	Congenital.	Acquired.	
Character	Often exceedingly loud, rough.	Usually soft, softer than in adults.	Always rough, scratching, scraping, sounding near the ear.
Localization	Slightly circumscribed, maximum point often not distinctly recognizable, or at the pulmonaris.	Slightly circumscribed, maximum point distinctly recognizable, situated at point of auscultation of a valve.	Audible only at narrowly limited space (although less circumscribed than in adults), usually at the base, sometimes at the apex of the heart.
Occurrence of systolic and diastolic action.	Systolic or diastolic, with a systolic murmur.	Systolic, or diastolic and systolic; almost never purely diastolic. Sounds usually not entirely covered.	Between systole and diastole; in no case regularly coinciding with either. Cardiac sounds audible.
Transmission	Strongly transmitted to the back, by arterial vessels rather to the upper parts, by venous to the lower, not to the arteries of the neck.	Partly strongly transmitted to the back; especially in great intensity, disturbance of compensation, and infiltration of the lungs.	Not transmitted.
Augmentation	By increase of the blood pressure.		By pressure on the thoracic wall at the point of auscultation and bending the body forward.
Decrease	By sinking of the blood pressure, at high frequency of pulse and respiration.		
Special qualities....	Often without frémissement, without change of cardiac dulness, and the cardiac impulse, and without accentuation of the second pulmonic sound.	In myocarditic murmurs of insufficiency findings are varying, otherwise rather constant.	Spontaneous in a short time, and varying upon change of position.
Accompanying and demonstrative circumstances.	Early appearance (not necessary). Often without hypertrophy and dilatation. Usually asthma and attacks of suffocation, drumstick fingers.	Cardiac asthma, digital deformities usually absent, hypertrophy seldom absent.	
Occurrence according to age.	Under 3 years.	Also under 3 years.	Seldom under 3 years.
Occurrence in pathological conditions, and etiological indications.	Congenital heart disease, transposition of the large vessels, open condition of the fetal circulation.	Acute, chronic, and ulcerative endocarditis. Acquired heart disease. Dilatation of heart, myocardiac muscular insufficiency. After infections: polyarthritis, tuberculosis, scarlet fever, sepsis; chorea; less frequently: typhoid, measles, varicella, erysipelas, influenza, diphtheria, pneumonia, gonorrhoea, osteomyelitis, mumps, syphilis.	Dry and exudative pericarditis. In and after the infections mentioned in previous column; also in affections of neighboring organs (spinal column, ribs, sternum, bronchial glands, lungs, pleura; caries, tuberculosis, pneumonia, pleuritis); in acute and chronic nephritis.

MURMURS

should happen to possess the rhythm of cardiac action in tachypnoea)

Cardiopulmonary.	Accidental = "anæmic."	By compression.	Pleuropericardial.
Rather rough, sometimes concealing the first sound entirely.	Soft, tender, blowing; never concealing the first sound; not loud.	Blowing.	Like the pericardial.
Moderately circumscribed; usually over the left ventricle, sometimes at the apex, quite seldom at the base of the heart or to the right side of the sternum.	Rather circumscribed; almost exclusively audible at the base (pulmonary), or at least there is the maximum point; never at aorta or tricuspid.	Base of heart, especially over pulmonary area.	Like the pericardial.
Always exclusively systolic.	Almost always systolic.	Systolic.	Like the pericardial.
Transmitted slightly or not at all.	Almost never transmitted to the back.		Not transmitted.
By inspiratory holding of breath, rapid heart action, excitement, low tension.			Partly increased by inspiration, partly diminished, often disappearing entirely on strong pressure.
Disappears with expiration and high tension and when breath is held.	In the dorsal position.		
.....	Rapidly changeable, especially as to intensity.		
Psychical excitement.	Constitutional disorders, cervical venous murmurs. Usually no change in cardiac dulness, no accentuation of the second pulmonary sound.		
Usually over 3 years, but exceptionally also earlier. In children much more frequently than in adults.	(Almost) never under 3 years, frequently at puberty.		
With rapid pulse and respiration, in excitable children with healthy hearts.	Nearly all forms of anemia of childhood, including leukæmia and chlorosis; highly febrile conditions; general disorders of cardiac nutrition.	Mediastinal tumors, pleuritic and pericarditic adhesions and cords; with soft thoracic wall on pressure of stethoscope; thoracic deformities.	Acute, chronic, and subacute pleurisy and peritonitis.

3. Dropsy and cyanosis of the skin (lips, nails!).
4. Oliguria, albuminuria, cylindruria (hyaline casts), hæmaturia (congested kidney).
5. Hydrothorax, hydropericardium, ascites.
6. Visible pulsation of the swollen veins of the neck, increased cardio-systolic collapse of the veins.
7. Chronic gastro-intestinal catarrh.
8. Chronic bronchitis (due to congestion).
9. Congenital and acquired valvular insufficiency in the state of non-compensation.—Chronic endocarditis and myocarditis; adhesive pericarditis; whooping-cough.

98. CHANGES IN THE NUMBER OF WHITE BLOOD CORPUSCLES

(For physiological data see Table belonging to No. 196. Counting after Thoma-Zeiss)

A. INCREASE, "LEUCOCYTOSIS."

1. *Physiological*.—Digestion, bodily effort; in the breast-fed child after the first feeding with cow's milk (Proportion of figures of the various forms unchanged). Attention should also be paid to the physiological diurnal variations.

2. *Pathological*:

(a) Neutrophilic leucocytosis (Increase refers particularly to the polynuclear neutrophilic leucocytes. Simultaneously in most cases eosinophile leukopenia).

(a) Certain blood diseases.—Leucocytic leukæmia (up to 70,000 per mm.*); of a moderate degree in splenic anæmia (up to 50,000) and pseudoleukæmia (slight), and simple anæmia (up to 20,000).

(β) Almost all acute intoxications and infections.—Especially pneumonia (Here continuously increasing until 2 days before the crisis), polyarthritis, diphtheria (up to 17,000), scarlet fever (up to 30,000), erysipelas, influenza, miliary tuberculosis, etc.; not existing, however, or existing only before (and at the beginning) of the eruptive period in typhoid, serum disease, measles, German measles, variola, vaccinia.

(γ) Almost all pathological conditions involving violent inflammation and formation of pus foci; e.g., appendicitis (20,000 and more), suppurative meningitis.

(δ) Certain constitutional diseases and cachectic con-

*In these and some of the following figures rare and extreme cases have been included.

A. INCREASE, "LEUCOCYTOSIS."—Continued

- ditions.—Rachitis, athrepsia, hereditary syphilis.
- (c) After hæmorrhages (for instance, in hæmorrhagic diathesis).
- (b) Eosinophile leucocytosis.—Leukæmia, nephritis, syphilis, tuberculosis, scarlet fever. Various dermatoses, intestinal parasites, [bronchial asthma, trichinosis].
- (c) Lymphocytosis (Increase refers particularly to the lymphocytes).—Simulated by the physiological relative lymphocytosis existing in the first few years of life. Whooping-cough, pernicious anæmia, tuberculosis, tuberculous meningitis, acute and chronic lymphatic leukæmia, gastro-enteritis of infants.

B. DIMINUTION, LEUCOPENIA.

1. Pernicious anæmia.
2. Measles and German measles, variola, vaccinia, serum disease after eruption (polynuclear leucopenia); typhoid in its entire course (sometimes even in inflammatory complications).
- [3. Banti's disease.]

99. CHANGES IN THE NUMBER OF RED BLOOD CORPUSCLES

1. *Increase, Hyperglobulia.*—Tuberculosis (?), loss of water (in diarrhœa, for instance), congenital heart disease with cyanosis.
2. *Diminution, Hypoglobulia, Oligocytosis:*
 - (a) Lymphatic leukæmia (down to 1.5 millions*, pseudoleukæmia, splenic anæmia (down to 0.8 million), pernicious anæmia (down to 0.25 million), and simple anæmia (down to 1 million).
 - (b) Some acute infectious diseases.—Diphtheria, polyarthritides.
 - (c) Many chronic (febrile) affections.
 - (d) Rachitis and infantile scurvy (moderate), athrepsia.
 - [(e) Hæmoglobinuria, after an epileptic attack, etc.]

100. CHANGES IN THE PERCENTAGE OF HÆMOGLOBIN

For demonstration, the apparatus and method of Fleischl are recommended.

(The Tallquist scale always resulted with us in higher comparative values. *For normal conditions see Table belonging to No. 196.*

1. *Increase, Polychromæmia.*—Pneumonia, diphtheria, tuberculous meningitis (prodromal), and chronic cardiac affections (here often with great pallor of the skin!).

* In these and some of the following figures rare extreme cases have been included.

2. *Diminution, Oligochromæmia.* (Simulated by pressing out lymph in obtaining the blood and other technical mistakes in determining the result!).

- (a) Splenic anæmia (down to 25 per cent.), simple anæmia (down to 10 per cent.), pernicious anæmia (down to 4 per cent.), leukæmia and pseudoleukæmia, chlorosis (down to 20 per cent. without hypoglobulia!).
- (b) Typhoid.
- (c) Rachitis, infantile scurvy (down to 40 per cent.), syphilis, Banti's disease.

SUPPLEMENTARY.

3. *Changes of the Histological Picture*:*

- (a) Occurrence of poikilocytes and anisocytes (of variously formed and different sized erythrocytes). (Simulated by mechanical errors in the preparation.)—Syphilis, rachitis, leukæmia, splenic and pernicious anæmia, congenital heart disease; seldom in infantile scurvy, chlorosis.
- (b) Occurrence of normoblasts and megaloblasts (nucleated red cells of normal and of abnormally large size). Syphilis, rachitis, myelogenous leukæmia, pernicious and splenic anæmia (seldom simple anæmia).
- (c) Occurrence of (eosinophile) myelocytes. Syphilis, myelogenous leukæmia, splenic anæmia, grave acute infections (diphtheria).
- (d) Occurrence of marrow cells (large mononuclear elements with neutrophile granulations). Myelogenous leukæmia.

4. *Changed Coagulability of the Blood:*

- (a) Increased.—Acute polyarthrits, pneumonia, athrepsia, light anæmia, pancreatic affections.
- (b) Diminished.—Typhoid, malaria, septic affections, pernicious anæmia, hæmophilia; inflammation of the kidneys, and fatty degeneration.

101. ACUTE ENLARGEMENTS OF THE LYMPH-NODES

(with inflammatory manifestations)

Simulated by development of lymphomata in acute lymphatic leukæmia and pseudoleukæmia (No inflammatory reaction, no adhesions with each other or with the skin, multiple formations). By parotitis (Localization! doughy consistency), submaxillaritis, inflammation of the submaxillary gland, periostitis.

* In order to obtain rapidly and conveniently a histological blood picture we have found Von Rzentkowski's method reliable. Make blood smears as usual on fresh, well cleaned slides. The air-dry preparations are immersed in olive oil, which is heated in a vial with a small flame to 117° F. The oil is removed by washing in ether (or benzoin). Stain with Ehrlich's triacid solution for 5-7 minutes.

1. Collateral lymphadenitis, limited to the glands situated in the region of a circumscribed pathological focus; inflammatory (suppurative) processes of a non-specific nature (demonstration of the primary pathological focus): stomatitis, angina, dental caries, retropharyngeal abscess, rhinitis, otitis, eczema, erythema, impetigo.

2. Lymphadenitis with and after general infections (Partly localized, partly general, frequently with circumnodular œdema). Scarlet fever, notably diphtheroid (Predominating in the neck; not infrequently suppurating, of long duration; sometimes only appearing after the subsidence of the fever in the third week, as in nephritis). Diphtheria (Much less frequently suppurating, in grave cases "pestilential" with "cou pro-consulair"). German measles (Especially behind the ear and at the occiput, also submaxillary, subangular, suboccipital, arranged in chains at the posterior border of the sternocleidomastoid, somewhat painful on pressure; prodromal signs!). Pfeiffer's glandular fever (Glandular tumors, rapidly developing under febrile manifestations behind the upper insertion of the sternocleidomastoid, later also at the occiput, up to pigeon-egg size, firm, elastic, very sensitive to pressure, without œdema of skin, at first unilateral, never suppurating, but always of long duration and very slowly receding; often pains in the neck, torticollis and after hæmorrhagic nephritis). Influenza, typhoid, parotitis, cerebral meningitis, erysipelas, somewhat pronounced with and after measles.

3. Lymphadenitis in toxic conditions (*e.g.*, snake bites) and serum disease (Regional at the point of injection; 7-8 days after injection, before onset of fever and exanthem; painful!).

LOCALIZATION OF ENLARGED LYMPHATIC GLANDS IN PATHOLOGICAL CONDITIONS IN THE BUCCAL AND FAUCIAL REGIONS.

Enlargement predominating in :	Seat of the Affection.
Submaxillary glands.	Malar mucous membrane, gums of the submaxillary bone, upper lip, tongue, malar integument.
Sublingual glands.	Mucous membrane of submaxillary bone and lower lip.
Pharyngeal glands.	Posterior middle parts of pharyngeal walls, part of interior of nose.
Lateral deep cervical glands (below and behind the sternocleidomastoid) and neck glands.	Upper portions of the pharynx, nasopharyngeal space.
Anterior cervical glands (in front of the sternocleidomastoid, at a level with the hyoid bone).	Tonsils and faucial pillars, deeper parts of the pharynx, laryngeal region, upper parts of the thyroid gland (secondarily also nasal and pharyngeal space).

102. CHRONIC ENLARGEMENT OF PALPABLE LYMPH-NODES

1. As part manifestation of constitutional affections: Scrofulosis (Glands firm, oval, up to hazel-nut size, mostly painless: manifestations in the organs of sense!). Status thymicus and lymphaticus (Chronic

hyperplasia of all lymph structures, also the lingual follicles, tonsils, spleen, and thymus; low stature of body with great fulness, appearance pasty and bloated, anæmia, chronic dermatitis). Rachitis (Only moderate degrees of swelling, glands mostly harder, dependent upon complicating conditions).

2. In chronic infectious conditions: Tuberculosis of lymphatic glands, with or without caseation (In the latter case often assuming the picture of pseudoleukæmia, except that there is fever, diazo reaction, tuberculin reaction; chronic enlargement of the supraclavicular glands with non-involvement of the cervical glands speaks for tuberculosis in the thoracic region). Hereditary syphilis (especially relapsing) (Small, hard, movable tumors; especially the epitrochlear flexure, behind the ear, in the neck, in the axilla, and groin). Chronic skin affections (with secondary suppurative processes of an obstinate nature).

3. After acute processes (*see No. 101*), sometimes persisting for a long time.

4. Chronic lymphatic leukæmia and pseudoleukæmia (Generally painless, multiple, without inflammatory manifestations, without adhesions to each other or to the skin, moderately hard, almost never suppurating, appearing first in the superior triangle of the neck; progressive; course findings in spleen and blood! Often tracheobronchial compression, *see No. 71*). Less frequently: splenic and pernicious anæmia and myelogenous leukæmia.

[5. True tumors of the lymphatic glands, lymphosarcomatosis (at first like pseudoleukæmia, later on infiltration of capsule, adhesion of the glands to each other and the surrounding tissues). Carcinoma.]

SUPPLEMENTARY: Micropolyadenopathia infantum (In cachectic children under 6 years of age; numerous glandular tumors in the neck, occiput, in the axilla and groin, the size of peas or small shot, very hard, round or oval, movable, not coherent, not painful on pressure, often arranged in chains, like strings of beads, along with hepatic and splenic enlargement). All cachexias of infancy, generally occasioned by tuberculosis (of various kinds and localization), chronic septicæmic gastro-enteritis, hereditary syphilis, chronic bronchial and pulmonary affections after infectious diseases, chronic pyodermatitis.

103. OLIGURIA AND ANURIA

Genuine, renal.—Defective secretion of urine in the kidney and its excretion (urination absent or infrequent, bladder slightly filled or empty, urine concentrated).

Simulated by dysuria and ischuria (bladder filled), *see No. 120*.

1. Loss of water of the whole body in general or loss by special calls for water.—Perspiration and diarrhœa, development of peritoneal and pleural exudates and transudates, and of œdema. Inanition (pyloric

stenosis, intestinal stenosis, vomiting of accumulated matter), cholera infantum, hyperthermia, heat stroke.

2. Changes in circulation (in nephritic affections, myocarditic debility, pericarditis, ingestion of cardiac poisons, such as ergot and digitalis), acute and chronic inflammatory processes in the kidney (except contracted kidney). (*See Table belonging to No. 109*).

3. Reflex inhibition of urine formation and excretion, as in traumatism, abdominal operations, grave and painful abdominal conditions of all kinds, phimosis, paraphimosis, constipation, urethral and ureteral affections, cystitis.

4. Neuroses.—Hysteria (With vomiting of urea-containing fluid).

5. Intoxications (also without pronounced nephritis); e.g., with benzol derivatives.

104. POLYURIA WITH POLLAKIURIA

Demonstration: In older children, collection of the daily quantity or measuring of the various portions. In younger boys, urine recepta-

FIG. 4a.



FIG. 4b.



Showing technic of obtaining urine in a small boy: a.—With adhesive plaster. b.—With a bandage.
(Prof. Pfaundler, *Children's Clinic at Graz*.)

cle of Raudnitz or improvised apparatus made by the aid of adhesive plaster or bandage and a small test tube. (See Illustration.) Similar provisions for little girls, or indirect determination by weighing cotton plugs or linen (placed in position) both before and after evacuation of urine. In practice, the quantity can frequently be only estimated. Other methods are those of Bendix, Hecker, and Lange.

Single specimens of urine are obtained either by catheterization or by placing the (small) children on a cold glass or porcelain vessel (urination on irritation by cold).

A. WITHOUT SIGNS OF (EXISTING) AFFECTION OF THE ORGAN.

1. Some conditions of hydræmia; simple and pernicious anæmia, and chlorosis.
2. Convalescence after acute febrile infections (typhoid, pneumonia).
3. Polydipsia (*see No. 21*) and habitual or occasional ingestion of much fluid.
4. Resorption of transudates, œdemas, and exudates.
5. Use of medicaments and articles of diet (diuretics, coffee, wine, beer, tea, mineral water).
6. Neuropathies (neurasthenia, hysteria, epilepsy, hemicrania, tetany, exophthalmic goitre, "arthritism" (Here frequently also pure pollakiuria, and beside the constitutional factor a special cause like psychic emotions, violent painful irritations, etc.).
7. Diabetes insipidus and mellitus.

B. WITH SIGNS OF AN AFFECTION OF THE ORGAN.

1. Contracted kidney and amyloid degeneration of kidney; less frequently, disturbances of circulation in kidney, pyelitis.
2. After relief of a temporary impediment to the urinary flow from the kidney (urinary calculi, kink of the ureters in floating kidney).
3. Affections of other abdominal organs (especially the pancreas), and particularly with lesion of the nervous centres in the abdomen (tuberculous peritonitis, tuberculosis of the mesenteric glands).
4. Affections of the central nervous system (meningitis, hydrocephalus, tumor, encephalitis).

105. ANOMALIES OF THE COLOR OF THE URINE AND OF THE URINE PIGMENTS

Aside from the changes occasioned by the different concentration of the normal infantile, urine is remarkably thin and light in healthy babies after the first few days of life.

1. *Dirty Blue Color* (occurring in purulent urine or on the addition of oxydizing agents, caused by indigo):

(a) *Indicanuria* (*i.e.*, increased excretion of indican):

Demonstration: Tests by Jaffe* and Obermayer (in the former test excess of calcium hypochlorite should be avoided, because it causes decolorizing. It is often sufficient to add crude hydrochloric acid to the urine at slight heat, in order to obtain the indigo blue, or indigo red).

*Urine mixed with equal parts of concentrated HCl and a few drops of liquor calcium hypochlorosi; chloroform shaken up with the colored mixture takes up blue pigment from it.

1. *Dirty Blue Color, etc.—Continued*

Urine of healthy young children exhibits indican only in traces, and that of the newborn or breast-fed infants, not at all.

Simulated by iodides in the urine when iodine is used, and by a pigment after the use of thymol and kindred substances.

(b) Stagnation of the gastric and intestinal contents, especially in the small intestine (intestinal stenosis, intestinal obstruction, peritonitic paralysis of the intestine,—chronic tuberculous peritonitis, appendicitis,—obstinate constipation).—Ulcerous conditions in the intestine (tuberculosis, typhoid), and gastro-enteritis, cachexia; pernicious anæmia.

2. *Yellowish Red Color* (Increased excretion of normal pigments (uroerythrin, urorosein) and pigments which are as yet unidentified.)—Before and after the excretion of albumin in cyclical albuminuria. Some febrile and many hepatic affections, chlorosis, cachexia.

3. *Brownish Red Color* (froth sometimes yellow), caused by (increased) urobilin:

Urobilinuria:

Demonstration of urobilin: Amyl alcoholic extract of urine shows green fluorescence on addition of ammoniacal alcoholic solution of chloride of zinc.

Small quantities of urobilin which cannot be demonstrated by this method are physiological (after the first few months of life in artificially nourished children).

Febrile diseases, especially nearly all acute infectious diseases; congested kidney; resorption of hæmorrhagic effusions; hepatic affections of almost all kinds (also after icterus from obstruction to the bile); dysentery, intoxications (especially blood poisons). Pernicious and splenic anæmia, chlorosis.

4. *Canary-Yellow to Beer-Brown (rarely Green) Color* (froth yellow, yellow deposit on filter caused by bile pigments, especially bilirubin.

Bilirubinuria:

Demonstration: Gmelin's test (adding to the urine less than its own volume of fuming nitric acid), Rosin's test (adding to the urine more than its own volume of attenuated tincture of iodine). In both cases the appearance of the green ring is the predominant and decisive factor. For occurrence, see *Icterus*, Nos. 198, 199.

5. *Bloody*. (Urine flesh water-red to blood-red, with a greenish glitter (bicolored); less often brown to mahogany color. Caused by blood pigment):

(a) Hæmaturia (see Nos. 117, 118).

(b) Hæmoglobinuria in consequence of hæmoglobinæmia:

5. *Bloody, etc.—Continued*

Demonstration of hæmoglobin, or methæmoglobin: Heller's test* (limit of sensitiveness: $\frac{1}{2}$ per cent. of blood). Should the urine not contain enough earth phosphates to produce a deposit, a quantity of the phosphates or of normal urine should be added. For aloin test and spectroscopic methods see special works.

Simulated by foreign pigments. In Heller's test these become attached to the solution.

(a) Grave infections, especially scarlet fever, typhoid, malaria, erysipelas, syphilis, diphtheria, Winckel's disease.

(β) Grave intoxications (chlorate of potash, carbolic acid, mineral acids, pyrogallie acid, naphthol, quinine, santonin, arsenic, fungoid poisons).

(γ) Extensive burns.

In all these cases, possibly also accompanying nephritis, as hæmoglobinuric nephritis.

(δ) Paroxysmal hæmoglobinuria (Chills, headache, cyanosis, anxiousness, often urticaria, œdema, some fever; passing rapidly).—Sometimes due to an occasional factor, like overexertion, influence of cold, or infectious diseases in constitutional disposition; but mostly syphilis.

6. *Various Strange Stains* after the use of medicaments (partly appearing only after having been exposed to the air for a time).

(a) Chrysophanic acid: Acid urine brownish yellow, alkaline urine purple (rhubarb, senna).

(b) Santonin preparations: Acid urine greenish yellow, alkaline urine red (semen cinæ, santonin cells).

(c) Carbolic acid, creosote and other tar preparations, folia uvæ ursi and other tannin preparations, antipyrin, resorcin, salicylic acid, naphthalin: Urine green, green-brown, or green-black.

106. ANOMALIES OF THE SPECIFIC GRAVITY OF THE URINE

Examination by urometer if possible, with mixed day urine.

A. URINE ABNORMALLY HEAVY.

1. High concentration. Loss of water, high temperature within and without the body; muscular work, diarrhoea formation of transudates; disturbances of circulation, and in inflammatory processes of the kidney; whooping-cough (1.022–1.035; in consequence of high concentration of the uric acid).

*Urine boiled with an equal part of solution of potash; the sedimented earth phosphates are stained red.

A. URINE ABNORMALLY HEAVY.—*Continued*

2. Abnormal (dissolved) constituents.—Diabetes mellitus (mostly 1.030–1.040), hæmaturia, hæmoglobinuria, pyuria, albuminuria (especially also intermittent, cyclical forms).

B. URINE ABNORMALLY LIGHT.

In low concentration.—Abundant ingestion of water, anæmia and renal affections with polyuria, diabetes insipidus, etc.

107. ABNORMAL ODOR OF THE URINE

1. Pungent, "urinous" (NH_3).—Ammoniacal cystitis. Rachitis (also after eating pickled herring), athrepsia.
2. Fruity (acetone).—Diabetes mellitus and other conditions of acetonæmia and acetonuria. (See No. 112.)
3. Various odors.—Violets: use of turpentine. Balsam: use of resins. Characteristic odors after ingestion of certain articles of nutrition and diet (*e.g.*, asparagus), and in poison cases.

108. ALKALINURIA

Simulated by decomposition of the urine outside the body (in rachitis and athrepsia apparently increased decomposability of the urine).

1. Caused by excess of fixed alkalies (Blued litmus paper remains blue when dried).—Physiological in the period of digestion of a liberal mixed meal, in uniformly vegetable diet, and in ingestion of carbonates and salt in large quantities.

Frequent evacuation of the gastric contents through vomiting or stomach washing in gastric disorders. Absorption of transudates, exudates, and blood (pneumonia 1–2 days after the crisis); hæmaturia and pyuria (pyelitis, nephritis, renal abscess).

2. Caused by production of ammonia (Blued litmus paper becomes redder again when dried).—Ammoniacal fermentation in the bladder, cystitis (except cholecystitis).

109. ALBUMINURIA

DEMONSTRATION:

PROCEDURE I: Fresh, cooled urine (cleared, if necessary, by filtration) is mixed with about one-eighth of its volume of concentrated nitric acid, and boiled.

TURBIDITY RESULTS.

Immediately.	Not until soon after cooling.	Not at all or only after a quarter of an hour.
True albuminuria (always pathological).	Albumosuria* (peptonuria), always pathological.	No albuminuria; no albuminoid substances or only mucin or mucinoid substances (behavior possibly physiological).

*Turbidity disappears again on heating.

PROCEDURE II: Urine as above (at very high concentration, diluted with one-half its volume of water) is mixed with traces of concentrated acetic acid.

TURBIDITY RESULTS.

Immediately; at the latest in 1 minute.		Not at all, or much later.
Turbidity on further addition of the acid.		Neither nucleo-albuminuria nor mucinuria.
Clears up.	Does not clear up.	
Nucleo-albuminuria.	Mucinuria.	

A few drops of a 10 per cent. solution of ferrocyanide of potassium are then added to the mixture.

TURBIDITY RESULTS, OR INCREASES.

Immediately.	After some time.	Not at all.
Albuminuria, in the strict sense.	Albumosuria.* (Peptonuria).	Neither albuminuria nor albumosuria.

Simulated by pyuria, spermaturia, menstrual hæmorrhage; high concentration of the urine, albumosuria; technically faulty chemical examination, and impure reagents.

TYPES

A. Occasional Albuminuria in individuals with no other pathological manifestations (No increased tension of pulse, no cardiac hypertrophy, no dropsy; no lasting granulated or epithelial cylinders, no renal epithelia; $A < 0.1$ per cent.; there is nothing but nucleo-albumin or mucin).—First 6–10 days of life, and the period of puberty; bodily (and mental) overexertion; after digestion of generous meals; effect of cold baths.

B. Cyclical Intermittent Albuminuria, occurring especially in certain constitutional anomalies (Usually purely orthostatic†; along with azoturia, increased excretion of urine pigment and urine salts; no elementary constituents).—"Neuro-arthritis" (in gouty, diabetic, and neuropathic hereditary tendency and together with signs of gouty disposition); cardiac and vesicular atony, functional heart diseases, chlorosis; chronic dyspepsia with gastro-intestinal atony. Scrofulo-tuberculous habit.

C. Albuminuria of Renal Origin, see Table between pages 144–145.

*Turbidity disappears again on heating.

†That is in erect posture.

110. MUCINURIA AND NUCLEO-ALBUMINURIA

Excretion of a protein substance in the urine which assumes a gelatin-like consistency on being mixed with acetic acid.—In many healthy children (especially the newborn), and after bodily exertion; in all catarrhal affections of the urinary ducts; in acute infectious and toxic conditions, with, before, and after renal albuminuria; in intermittent albuminuria, urolithiasis, congested kidney, etc.; and also in catarrhal icterus, leukæmia.

111. ALBUMOSURIA, PEPTONURIA

Demonstration, *see* No. 109.

1. Pyogenous form.—Absorption of cellular or suppurative exudates in pneumonia (stage of resolution), pleuritis, meningitis, polyarthritides, pyæmia, abscesses of bones, skin, and glands, etc.
2. Infectious form.—Typhoid, scarlet fever, erysipelas, diphtheria, measles, mumps, malaria.
3. Hæmatogenous form.—Blood effusions of all kinds into the tissues. Leukæmia, purpura.
4. Hepatogenous form.—Hepatic processes of various kinds.
5. Renal form.—Precursor of renal albuminuria.

112. ACETONURIA

Demonstration: Legal's test* (solution of soda should be added drop by drop as long as the color visibly increases; excess should be avoided).

Traces of acetone physiological; demonstrable quantity in inanition, increased tissue degeneration, and insufficient oxidation under certain conditions.

1. Diabetes mellitus (large quantities!).
2. Acute gastro-intestinal affections with acetonæmia and possibly eclampsia or tetany (gastro-enteritis in nurslings, cyclical vomiting in older children).
3. Highly febrile, acute infections, especially croupous pneumonia, scarlet fever, measles (stage of eruption); (much less frequently non-infectious or afebrile infectious diseases).
- [4. A few ectogenous intoxications: Phosphorus, lead; anti-pyrin.]
5. Deficiency of oxygen in asthma accompanied by laryngeal stenosis, narcosis.

*Urine mixed with about one-fifth of its volume of fresh, concentrated, aqueous solution of sodium nitroprusside, and a few drops of solution of caustic soda; upon addition of an excess quantity of acetic acid there occurs a burgundy-red color.

113. DIACETURIA

Appearance of aceto-acetic acid (*i.e.*, diacetic acid) in the urine. (Always associated with acetonuria.)

Demonstration: Bordeaux-red inconstant coloration on adding a solution of chloride of iron drop by drop to (fresh!) urine. Test negative in the boiled urine, positive in acid ethereal extract from fresh urine.

Simulated by the (somewhat deviating) shade of urine containing salicylic acid or antipyrin.

Occurs under same conditions as acetonuria.

114. GLYCOSURIA

Occurrence of grape (and other) sugars in the urine in quantities which are demonstrable by the usual methods.

Demonstration: Reduction tests (Trommer, Worm-Müller*, Böttger); in the case of doubtful reaction, also fermentation test (in bacteriological fermentation flasks), polarimetric examination, production of a glucosazone. Milk sugar not fermentable.

Physiological in ingestion of sugar beyond the limit of assimilation (see 2, (a), below), [and in the healthy newborn?].

Simulated by technical mistakes in the chemical examination, by the presence of much uric acid, creatinin, glycuronic acid, chrysophanic acid (use of rhubarb).

1. Diabetes mellitus (Generally constant, very abundant sugar, up to 8 or 10 per cent.).

2. Glycosuria (Occurring only periodically; usually little sugar):

(a) Alimentary after feeding with sugar or starch; reduction of the limit of assimilation (as part manifestation of hepatic disorders, especially in hereditary syphilis, dyspepsia, intoxication, exophthalmic goitre, and other conditions of hyperthyroidism).

(b) Toxic (phloridzin, opium, morphine, chloral, alcohol, amyl nitrite, turpentine, carbon-dioxide, prussic acid, sulphuric acid, mercury).

(c) Acute infectious diseases (typhoid, scarlet fever, diphtheria, sepsis, cerebrospinal meningitis, [whooping-cough]).

(d) Affections of the central nervous system and neuroses: After epileptic and apoplectic seizures, cerebral tumor, encephalitis, spinal meningitis, exophthalmic goitre, tetany, sciatica.

(e) Affections of abdominal organs, especially pancreas and liver; also in dyspepsia, athrepsia.

*Sedimentation of the separated protoxide of copper in the centrifuge considerably increases the delicacy of the test.

C. Renal albuminuria.	Character of Urine.				Foreign and	
	Appearance.	Daily quantity.	Specific gravity.	Amount of albumin.	Casts.	Blood.
1. <i>Albuminuria in Disturbed Circulation of the Kidneys:</i>						
(a) Active, hyperemia.	Clear, somewhat darker.	Reduced, may be increased.	Increased.	Slight (often only albumosuria).	Very few; hyaline.	Occasional erythrocytes
(a) Acute, febrile infections, especially typhoid, diphtheria, and pneumonia (second to tenth day of illness), scarlet fever, polyarthritis, measles, influenza, especially in hyperthermia.						
(β) Intoxications (qualitative, like 3, (a) (γ)).						
(γ) Accompanying other nephritic affections (tumors, lithiasis, tuberculosis).						
(b) Passive hyperemia, congested kidney of the first degree.—Valvular insufficiency, myocarditis, heart dilated, chronic infiltration of the lungs, whooping-cough.	Dark.	Usually small.	Increased.	Slight (dependent on condition of the heart. Digitalis medication very often reduces the albuminuria).	Hyaline.	Occasional erythrocytes
(c) Other disturbances of the circulation (not exactly known, retardation of the circulation, lowering of blood pressure?).		Changeable, usually increased.	Changeable.	Usually slight; varying.	Hyaline; very few.	Usually none, or a few erythrocytes. Exception: leukemia.
(a) Anemia, leukemia, pseudoleukemia.						
(β) Functional and organic neuroses (epilepsy, apoplexy, hysteria, chorea, meningitis, tetanus). Here may also belong albuminuria of serum disease and partly also the "orthostatic forms of albuminuria."						
2. <i>Albuminuria in Degeneration of the Kidneys:</i>						
(a) Parenchymatous and fatty degeneration.—Effect of injuries, like 1 (a) and 3, for a long-continued time.				Slight (often only albumosuria).	Hyaline; few.	None.
(b) Amyloid degeneration.—Tuberculosis, syphilis, malaria, dermatosis, especially purulent skin affections.	Pale, light, clear.	Somewhat increased (diminished just before death).	Usually reduced; not constant.	May be very great; sometimes slight, however.	Few; wax-like, hyaline, and granulated.	Usually none.
3. <i>Albuminuria in Acute Nephritis:</i>						
(a) Acute parenchymatous nephritis.						
(a) Catching cold, getting wet.						
(β) Acute infections, especially scarlet fever, diphtheria, measles, typhoid, whooping-cough, pneumonia, influenza, Pfeiffer's glandular fever, varicella, mumps, pyemia, sepsis, erysipelas, vaccinia, dysentery, malaria, morbus Weilli, non-specific angina, stomatitis, enteritis, appendicitis, cystitis, etc. [and chronic infections: hereditary syphilis].	Often flesh-water color; pale red to dark red; cloudy.	Greatly diminished, sometimes nothing; at the same time vesical tenesmus.	Greatly increased; to about 1.030.	Great, up to 1 percent. (rarely slight or almost none).	Abundant; especially blood and epithelial casts, granulated and hyaline.	Often abundant; sometimes also hemoglobinuria.
(γ) Intoxications (also articles of nutrition and medication), especially cantharides, tar, iodine, turpentine, balsam of Peru, carbolic acid, salicylic acid, sulphuric acid, calcium chloride, mercury, alcohol.						
(δ) Pyoderma (like impetigo, pemphigus).						
(b) Acute exacerbation of chronic nephritis.	Same.	Usually reduced, but only moderately.	Not materially increased.	Same.	Same, also metamorphic casts.	Same.
(c) Acute suppurative nephritis.—Cystopyelitis, nephrolithiasis, septicopyemia, after tumors, renal tuberculosis.	Very turbid.	Normal.	Normal.	Moderate; but more albumin than corresponds to the quantity of pus (1 mg. albumin = 80,000 L.).	Usually none, never abundant.	May be present.
4. <i>Albuminuria in Chronic Nephritis:</i>						
(a) Chronic parenchymatous nephritis.—Chronic Bright's disease; special form: chronic hemorrhagic nephritis. Injuries with continued effect as mentioned under 3, (a) (especially also syphilis, tuberculosis, chronic gastro-enteritis), or later stage of 3, (a).	Flesh-water color or dirty yellow.	Changeable, but usually diminished.	Normal or somewhat increased.	Abundant.	Abundant; all kinds, especially also fatty.	Usually present.
(b) Chronic interstitial nephritis.—Contracted kidney, granular atrophy. Syphilis, diabetes, lead, alcohol.]	Pale, clear yellow; green-yellow.	Greatly increased, pollakiuria.	Greatly reduced.	Slight, at times almost none.	Very few; hyaline.	Occasional erythrocytes
(c) Heubner's "dubious forms."		Normal or reduced.	Normal or increased.	Slight, also intermittent.	Few; hyaline, rarely granulated.	None.

*Uremic manifestations, including the following: anorexia, nausea, vomiting, diarrhoea, intermittent respiration, asthma, vertigo, &

OF RENAL ALBUMINURIA

Substances in urine.			Albuminuria accompanied by				Course of the manifestation.	Other concomitant manifestations.
Leucocytes.	Epithelia.	Chlorides and phosphates.	Uræmia.*	Heart and pulse (consecutive changes).	Edema and ascites.	Retinitis albuminurica.		
None.	Very few.	Normal.	None.		May be present in traces.	None.	Passing rapidly.	
None.	None.	Normal.	None.	Affected by the primary process.	Present, but dependent on the primary affection.	None.	Changeable, intermittent.	Signs of engorgement in general circulation, pulmonary or cardiac affection, sinking of the blood pressure. Urea diminished urobilinuria.
			None.	Usually none.	Sometimes present.	None.	Long-continued, changeable.	
	None.		None.		Absent.	None.		
	Few.	Reduced.	Seldom or never.	No hypertrophy.	Distinctly present.	None.	Chronic progressive.	Never in the youngest children! signs of other amyloid degenerations (spleen, liver, intestine). Urea and uric acid normal.
Abundant.	Abundant (also fresh).	Greatly reduced.	Frequently.	Usually no hypertrophy; great tension of pulse.	Usually present, but may be absent; edema, changing greatly; (ascites especially strong after scarlatina).	None.	Acute.	Pain (fever may be absent). Urea diminished. Uric acid normal.
Same.	Fatty.	Same.	Same.	Hypertrophy.	Same.	Present.	Chronic, exacerbating.	Manifestations of chronic renal affection.
Often in large quantity.	Abundant; often entire tissue shreds.	Reduced.	Ammonismia.			None.		Fever, pain, tumor; often cystitis, alkalinuria.
Present.	Present.	Reduced.	Frequently.	Always distinct hypertrophy.	Very distinct, sometimes excessive (edema especially in scrotum and lower extremities).	Often present.	Subacute or chronic.	Chronic hemorrhagic form always exacerbating. Paroxysmal exacerbation of the urine and the general condition. Tendency to inflammatory affection of mucous and serous membranes and to hemorrhages. Dyspepsia. Urea and uric acid diminished.
None.	Very few.		Very frequently.	Distinct hypertrophy never absent; wire pulse.	Moderate or absent; usually only with occurrence of cardiac insufficiency.	Often present.	Very slow, creeping.	
Abundant.	None.		None.	No change.	Usually absent.	None.		

numbness, delirium, sleeplessness, coma, headache, spasms, amaurosis, tinnitus aurium, and pruritus.

115. DIAZO REACTION IN THE URINE

Demonstration: Ehrlich's test*, simultaneously observing the secondary and tertiary reactions.

Simulated (the primary and secondary reactions, not the tertiary) in the use of medicaments, especially phenol and its derivatives (creosote, guaiacol, also thymol, phloridzin, opium, cascara, hydrastis, santonin, and rhubarb). Demonstration interfered with through tannin medication.

Occurrence general, especially in transmission of bacteria (or their metabolic products?) into the blood.

1. Severe acute infections: Measles, typhoid, acute miliary tuberculosis (in these practically constant). Scarlet fever, severe croupous pneumonia and cerebrospinal meningitis, sepsis with ulcerous endocarditis. [Erysipelas, polyarthritis, varicella, non-specific angina, malaria, influenza?]

2. Tuberculosis, taking a chronic course. Especially chronic cavernous pulmonary tuberculosis with hectic fever; less frequently in chronic miliary tuberculosis of various organs, caseous pneumonia, pure pleuritis, peritonitis, caries, tuberculous meningitis, and tuberculosis of the lymphatic glands, which runs under the picture of pseudoleukæmia.

[3. After chloroform narcosis, in severe disturbances of compensation, in insufficiency of the cardiac valves, in agony.]

116. OCCURRENCE OF ORGANIZED URINARY SEDIMENTS

Demonstration by microscopical examination of the sediment obtained by the centrifuge (possibly also in the beaker), or of the filter residue. Many cellular ingredients become recognizable only by staining (addition of a little Lugol's solution or methylene blue). Undesirable inorganic salts and excess of staining substances can be appropriately removed by alternate decanting, washing with water, and centrifuging.

1. *Formed Mucus* (Cylindroids, very long, of muco-filamentary construction, varying in thickness, ligamentous; sometimes—in ammoniacal reaction of the urine—gelatinous masses).—Cystitis, pyelitis, nephritis, renal tuberculosis.

2. *Blood Elements* (see *Hæmaturia*, No. 117).

3. *Pus Corpuscles: Pyuria* (Simulated by inorganic sediments: urates, phosphates):

(a) Considerable pus.—Severe cystitis (in myelitis; severe acute infections, tuberculosis, gonorrhœa; severe gastro-intestinal conditions, especially colitis, appendicitis; poison-

*Concentrated, weak hydrochloric solution of sulphanilic acid mixed with a few drops of a 1 per cent. solution of sodium nitrite, then with an equal volume of urine. Addition of NH_3 gives red coloration (secondary reaction); on allowing the mixture to stand, a ring-shaped green deposit is formed (tertiary reaction).

3. *Pus Corpuscles: Pyuria, etc.—Continued*

ing by cantharides; catheterism, pollution, foreign bodies and urinary calculi). Perforation by perinephritic, pericystitic, perityphlitic, perimetritic abscesses; hydro-nephrosis.

- (b) Small or moderate quantities of pus.—Cystitis, urethritis, pyelitis (Distinctly cylindrical arrangement of the pus cell masses, no true urinary casts, rapidly changing condition of the urine—in uracidosis, balanitis, gonorrhœa, nephrolithiasis, tuberculosis), suppurative nephritis, kidney infarction in severe intestinal disorders, renal tuberculosis, renal tumors.

4. *Fat Droplets: Lipuria* (simulated by fat in lubricant on catheter).—Fatty degeneration of the kidneys.

5. *Epithelia:*

(a) Pavement epithelia from the vulva.—Vulvitis.

(b) Tailed, imbricated, and conical, lamellated, multiform epithelia from the excretory urinary ducts.—Cystitis, pyelitis; also affections of the renal pelvis.

(c) Small, polygonal or rounded-quadrilateral epithelia, with very granular cell body plasma and distinctly visible, large oval nucleus, renal epithelia.—Disturbances of circulation, degeneration and inflammation of the kidney.

6. *Shreds of the Mucous Membrane and Pseudomembranes.*—Diphtheritic cystitis, tuberculosis of the bladder and kidney.

7. *Casts: Cylindruria:*

(a) Without albuminuria in healthy kidneys.—[Healthy newborn.] Hepatogenous icterus, [general tuberculosis and diabetes mellitus].

(b) With albuminuria in affected livers:

(a) Hyaline casts (Size greatly varying, homogeneous consistency, therefore difficult to see, finely marked).—Disturbed circulation in the kidney, degeneration and inflammation. Uric acid infarction in the kidneys of the newborn.

(β) Waxy casts (Similar to above, but with a dull lustre, yellowish, often with amyloid reaction).—Various chronic renal affections.

(r) True granular casts, with stratifications of blood and epithelia.—Especially in acute parenchymatous, hæmorrhagic and suppurative nephritis; also, renal tuberculosis.

8. *Bacteria.*—Of importance especially: bacteria of the colon and proteus groups in acid cystitis; micrococcus and bacillus ureæ in alka-

line cystitis; tubercle bacilli, typhoid bacilli; gonococci, erysipelas cocci, pyogenic bacteria of various kinds.

117. HÆMATURIA

Demonstration of the blood pigment, *see No. 105*; demonstration of the cellular elements of the blood by the microscope.

Simulated by premature menstruation; by hæmoglobinuria in a purely chemical examination; appearance may be simulated by excretion of chrysophanic acid (decolorized on addition of acid), (ingestion of rhubarb and senna), and black discoloration of the urine after ingestion of phenol derivatives, in indicanuria, etc.

Laymen are apt to describe highly concentrated (fever) urine as containing blood.

A. IN THE NEWBORN: Causes of hæmorrhage (*see No. 63*).

B. IN OLDER CHILDREN:

1. *Blood Quite Fresh, Unchanged, only at the Beginning or at the End of Micturition in the Shape of Tubular, Ligamentous, or Lumpy Coagula.*—Hæmorrhage from the lower urinary ducts, urethra, or bladder:
 - (a) Partial manifestation of a hæmorrhagic diathesis or blood disease: hæmophilia, purpura hæmorrhagica, infantile scurvy, leukæmia, pseudoleukæmia, pernicious anæmia, septic infections (scarlet fever, severe diphtheria, sepsis, ulcerative endocarditis).
 - [(b) Urolithiasis.]
 - (c) Cystitis and severe pyelitis.
 - [(d) Tumors (Findings in successive portions very variable; blood often abundant, pain on micturition).]
 - (e) Traumatism (catheter, pelvic fracture).]
2. *Blood Fresh or Discolored, Uniformly Mixed with Every Portion of the Urine; Blood Casts and Urinary Casts simultaneously.*—Hæmorrhage from the kidney (including the renal pelvis).
 - (a) Partial manifestation of a hæmorrhagic diathesis (*see 1, (a) above*). [Also cyclical, occurring in paroxysms, in relation to acute infections with or without succeeding nephritis: renal hæmophilia.]
 - [(b) Nephrolithiasis.]
 - (c) Acute and chronic (exacerbating) hæmorrhagic and suppurative nephritis (Urine generally "flesh-water-colored"; erythrocytes as "shadows" or rings).
 - (d) Renal tumors (Periodic hæmorrhages).
 - (e) Traumatisms.
 - (f) Renal tuberculosis (almost always tuberculosis in other organs).

B. IN OLDER CHILDREN.—Continued

- (g) Greatly congested kidney (Usually only isolated blood corpuscles) and hæmorrhagic kidney infarction (sudden onset) in cardiac affections.

118. SYNDROME: PYURIA, HÆMATURIA, AND NEPHRALGIA

1. Suppurative nephritis, renal abscess (Pus fever, chills, ammoniæmia).
2. Renal tuberculosis (Bacilli in urine—attention to be paid to smegma bacilli!; other signs of urogenital tuberculosis: testicles, ovaries, Fallopian tubes).
3. Renal tumor (*see No. 123*).
- [4. Nephrolithiasis (Concretions in fresh urine, colicky pains).]
- [5. Hæmorrhagic kidney infarction in cardiac affections (Sudden onset, rapid disappearance).]
- [6. Renal venous thrombosis in cholera infantum and cachexia of nurslings.]

119. OCCURRENCE OF NON-ORGANIZED URINARY SEDIMENTS

Demonstration by sedimentation (centrifugation) of the clean, fresh urine; microscopic and chemical examination of the sediment.

1. Uric acid (in freshly evacuated urine).—In the newborn, physiological; in older children, occasionally during fever. Habitual, suspicion of uric acid diathesis.
2. Urates (in freshly voided urine).—In high concentrations and in the first days of life, normal; should the urine be thin and pale, suspicion of urolithiasis and arthritism (Often also large sandy deposits).
3. Calcium oxalate (Gray, sandy masses) after partaking of certain fruits and vegetables (apples, pears, cauliflower); in diabetes mellitus, catarrhal icterus, and various other conditions.
4. Triple phosphate and ammonium urate (White granular masses of characteristic microscopic appearance).—Ammoniacal fermentation of the urine.
5. Large concretions of uric acid, phosphates, and oxalates.—Urolithiasis.
6. Leucin and tyrosin.—Acute phosphorus poisoning and acute yellow atrophy of the liver.
7. Bile pigment crumbs.—Icterus neonatorum.
8. Cystin.—Intestinal disorders.

120. DYSURIA, ISCHURIA, RETENTION OF URINE

Abnormal (especially difficult), painful urination, and retention of urine. Retention physiological: Healthy newborn babies sometimes void no urine for 2 days after the first bath (bladder empty!).

A. IN THE NEWBORN.

1. Congenital malformation of the external urinary passages: Diverticula and retention cysts, cloaca; epithelial atresia of the urethra, pathological increase of the (physiological) agglutination between the glans and the interior lamella of the prepuce; adhesion of the labia minora.
2. Occlusion of the urethra by concretions of uric acid; spastic conditions of the neck of the bladder in consequence of irritable condition and high percentage of uric acid in the urine.

B. IN NURSLINGS AND OLDER BOYS (aside from possible persistence of the above-named conditions).

1. *Direct Mechanical Organic Causes:* Phimosis, paraphimosis, prolapse of the urethral mucous membrane; tumors of the bladder, considerable distention of the abdomen, constipation, sclerœdema at the genitals; urolithiasis (Periodical pains, mechanical disturbances of micturition, interruption of stream on change of position; symptoms of cystitis; prolapse of the rectum; urinary concretions; after catheter examination!).
2. *Caused by Disorders of Innervation:* Paresis of the detrusor, spasm of the sphincter, inability to innervate voluntarily the former or to relax the latter, reflex disturbances:
 - (a) Organic changes of the innervation apparatus.— Transverse myelitis, spondylitis, tubercular meningitis, spinal leptomeningitis, polyneuritis (nervi pudendi).
 - (b) Participation in general disturbances of innervation.— Tetany, tetanus.
 - (c) "Reflex inhibitions" (especially frequent in constitutional neuropathy: neuro-arthritis, hysteria).
 - (α) In affections of the external urinary ducts: balanitis, balanoposthitis (varicellar vesicles!, etc.), vulvitis, vulvovaginitis (*see No. 126*), urethritis (urinary sand, gonorrhœa, masturbation), cystitis, [pyelitis].
 - (β) In other affections, especially those of the intestine: pelvic appendicitis, intussusception, peritonitis, intestinal parasites, hæmorrhoids, anal fissure, tenesmus.
3. The gravest general conditions, accompanied by disturbed consciousness, coma, collapse, third stage of meningitis, idiocy.

121. VESICAL TENESMUS

1. Every kind of polyuria (*see No. 104*).
2. Increased irritability of the mucous membrane of the bladder and irritating condition of the urine.—Vulvovaginitis, prolapse of the urethra, cystitis, acute nephritis, urolithiasis, oxaluria.
3. Spinal affections: Spinal leptomeningitis, and others.

122. INCONTINENCE OF URINE, ENURESIS NOCTURNA

Physiological while asleep up to the age of about 24 months; while awake up to the age of 10 to 12 months.

	With organic changes in the region of the genito-urinary tract.	Without organic changes of the genito-urinary apparatus.
Habitual condition.	1. Defective closure of the urinary ducts. Ectopia vesicæ, hypospadiæ, epispadiæ, congenital defective development of the sphincter vesicæ. 2. Stenotic factors: Permanent stricture of the urethra, congenital stenosis of the external urethral orifice, phimosis, agglutination of glans and prepuce. 3. Hypoplasia of the genito-urinary apparatus (?).	1. Habitual psychic defects.—Bad habits, laziness, defective education, idiocy, myxœdema, mongoloid, hydrocephalus, arthritis, degeneration (adenoid vegetations), hysteria, infantilism. 2. Absolute and relative insufficiency (atony) of the sphincter.—General debility, rachitis, anemia, spina bifida (occulta), constipation with habitual straining. 3. Increased reflex excitability of the detrusor muscle (together with factors producing irritation, such as violent excitation of a sensitive peripheral region, intestinal parasites, masturbation). 4. Changed consistency and increased quantity of the urine.—Diabetes mellitus, diabetes insipidus, increased acidity of urine.
Accidental condition.	Vulvovaginitis, cystitis, tuberculosis of the urinary system, cystic tumor.	1. Disturbance of consciousness, with severe general condition and cerebral affections.—Cerebral pneumonia, third stage of meningitis, cerebral tumor, epilepsy, eclampsia (paroxysm or "equivalent," often before the advent of the actual convulsions; early symptom), coma, apathy, agony, pathologically deep sleep. 2. Absolute and relative insufficiency of sphincter, with general debility, paralysis of sphincter and increased straining.—Transverse dorsal and cervical myelitis, traumatic myelitis (Pott's disease), [anterior poliomyelitis,] infantile tabes, spinal syphilis, tumors of the spinal cord, hæmorrhages of the spinal cord, postdiphtherial paralysis. Convalescence after grave affections. Whooping-cough and laryngospastic attacks. 3. Increased reflex excitability of the detrusor muscle and sensitive irritations at the periphery (as, for instance, anal fissure, eczema of the anus). 4. Disturbances of coördination, chorea minor; juvenile ataxia.

123. ENLARGED KIDNEY

Immovable either by respiration or by hand, always covered by the colon; the latter pushed forward, protruding in relief at the abdominal wall; frequently signs of functional renal insufficiency, changes in the urine, lumbar pains, neuralgia of the abdominal wall.

Demonstration by bimanual palpation. The normal kidney is rarely palpable.

Simulated by: Floating kidney (Considerable mobility and spontaneous change of position, characteristic shape and size). Tumors of

the mesenteric and retroperitoneal glands (Less regular, knotty, usually situated more toward the median line, and without change in the urine). Impaction of feces (voidable), ascites (symmetrical, movable dulness), inflammatory infiltration of the peritoneum and omentum, encysted peritoneal exudate, psoas abscess, hepatic tumor (Movable by respiration, usually affecting both lobes, thoracic walls bulging out). Enlarged spleen (Can be grasped, situated over the colon, movable on respiration, shape of spleen). [Ovarian cysts.]

1. Sarcoma and carcinoma (Especially in male children of the first and second years, rapidly growing, knobby, situated between iliac crest and costal arch, with pseudofluctuation; pain, cachexia, œdema, ascites, periodical hæmaturia).

2. Hydronephrosis (Fluctuating, globular, smooth, tense, growing slowly, no fever, no pains; condition and evacuation of the urine often normal—from the healthy kidney!; sometimes oliguria, uræmia, pyuria, hæmaturia, cylindruria).—Congenital bilaterally, acquired after stenosis of the excretory urinary ducts (calculus, tumor, floating kidney).

3. Perinephritis (Very painful, usually no urination, fluctuation, rapid development, pus, fever, and no vomiting).—Traumatic, after infectious diseases, vertebral caries, pyelitis.

[4. Thrombosis of the renal veins in sepsis, etc. (echinococcus of the kidney.) Renal tuberculosis.]

124. URÆMIA

For syndrome, see *Table Renal Albuminuria, belonging to No. 109*. Simulated by constipation, appendicitis, intoxication, meningitis.

1. Acute and chronic parenchymatous and interstitial nephritis.

2. Renal tumors, hydronephrosis, nephrolithiasis, renal degeneration.

3. Cystitis and all other inflammatory affections of the urinary passages.

4. Phimosis and other stenotic affections of the urinary passages.

5. Cyclical albuminuria.

125. TUMORS OF THE SCROTUM OR OF THE INGUINAL CANAL

1. Inguinal hernia (Tumor reducible with a gurgling sound, soft, oval, not demarcated, non-transparent, usually unilateral (right); percussion generally elicits intestinal sound. Testicles in normal place, inguinal ring open).—Especially in phimosis, constipation, whooping-cough, rachitis, urolithiasis; always congenital.

2. Hydrocele of the tunica vaginalis (closed) (Tumor often bilateral, developing in the first few weeks of life, not reducible, incompressible, oval, fluctuating, slightly tense, transparent, smooth, not reaching into the abdomen; spermatic cord normal, testicles only indistinctly palpable at the side or not at all).

[3. Hydrocele of the tunica vaginalis (open) (As in 2, above, but can be squeezed out toward the abdomen, seminal cord thickened; congenital.)]

4. Hydrocele of the funicular process (closed) (Tumor not replaceable, fusiform, fluctuating, very tense, rather hard, testicles free in normal place).

[5. Communicating hydrocele of the funicular process (As in 4, above, but reaching into the abdomen and may be reduced; seminal cord thickened).]

6. Cryptorchis (Tumor not reducible, roundish, hazelnut-size, smooth, elastic, remaining in the inguinal canal, non-transparent, scrotum empty).

7. Orchitis (and epididymitis).—Mumps (Fever, pain, infiltration of the scrotal integument; almost never under 8 to 10 years of age). Syphilis (Painful tumor, often hardening and obliteration of gland with functional derangement, infantilism, etc.). Tuberculosis (Enlargement of the testicles and epididymis, adhesion to the scrotal integument, perforation, evacuation of yellow-green pus, usually left). Influenza. After traumatism.

[8. Tumors of the testicles: Carcinoma and sarcoma.]

9. Œdema, phlegmon, and gangrene of the scrotum.—Erysipelas, contusion, general cedema.

126. DISCHARGES AND ABNORMALITIES OF THE VULVA

A. "FLUOR ALBUS."

1. Desquamative catarrh in newborn children (otherwise healthy children show at the introitus vaginæ a mucocreamy substance, consisting in the main of epithelial remnants).
2. (Simple) vulvovaginitis (of non-specific nature) (Œdema, hyperæmia, mucous secretion, here and there minute ulcers, collateral lymphadenitis, easily curable, terminating favorably).—Masturbation, oxyuria, pruritus, anæmia, chlorosis, scrofulosis, diabetes, foreign bodies, dermatosis in the neighborhood, tuberculous peritonitis.
3. Vulvovaginitis accompanying acute exanthemata (often prodromal): Scarlet fever, measles, varicella, variola, vaccination.
4. Gonorrhœal vulvovaginitis (Almost always with urethritis, sometimes with involvement of the cervix; flow at first thick, yellowish green, creamy-purulent, later sero-mucous; very obstinate course; seldom development of pointed condylomata; usually disturbance of the general condition).

A. "FLUOR ALBUS."—*Continued*

5. Syphilitic vulvovaginitis (Rarely primary condition; broad condylomata in symmetrical flat pictures).
6. Membranous vulvovaginitis, diphtheria of the vulva (Strongly attached coherent membranes, leaving behind ulcers with malodorous secretion, nearly always secondary after faucial attacks in debilitated children; Löffler bacilli).—Similar non-specific conditions in typhoid, scarlet fever, aphthæ.
7. Phlegmonous and gangrenous vulvovaginitis (Of the labia majora mostly unilateral discolored swelling, fetid odor, with deep ulcers).—Erysipelas, diphtheria, scarlet fever, noma, typhoid, tuberculosis.

B. HÆMORRHAGIC DISCHARGE.

1. Hæmorrhages in the newborn from the vagina (Otherwise healthy children show slight hæmorrhage and usually simultaneous swelling of the mammary glands from the first to the fifth day of life).
2. Premature menstruation, exceptionally between the first and seventh year.—Tuberculous hereditary tendency (?), obesity (?); in older girls metrorrhagia (dyscrasia, infectious diseases, heart and liver disorders).
3. Vulvovaginitis of specific or non-specific nature in erosions and ulceration through maceration, in vulvar tuberculosis (Irregular ulcer on the labia minora).
4. Prolapse of the urethral mucous membrane (In the orifice of the urethra a dark red, easily bleeding ridge, through which the catheter enters the bladder; sometimes swollen and œdematous.—Affections of the mucous membrane).
5. Hæmorrhagic diathesis.
6. Papillomata and other tumors.

127. JACTITATION, DELIRIUM

Simulated by chorea.

1. Many acute infections: Fulminating scarlet fever, typhoid, erysipelas, influenza, dysentery, croupous pneumonia, sepsis, miliary tuberculosis, anterior poliomyelitis, ulcerative endocarditis, and other hyperpyretic conditions.

2. Intoxications: Endogenous—cholæmia, uræmia, pyotoxæmia, dyspnœa. Ectogenous—alcohol, salicylic acid, digitalis, coffee, tea, alcohol, arsenic, iodoform.

3. Disturbances of circulation in the brain: Anæmia, cerebral hyperæmia, sinus thrombosis.

4. Meningitis of all kinds.

5. Paralysis of the heart.—Vagus affection, myodegeneration.

128. TREMOR

Tremor of the submaxillary bone physiological in the newborn.

1. Organic affections of the cerebrospinal system (meninges, cerebrum (especially the cerebellum), spinal cord) and of the peripheral nerves: Chronic hydrocephalus, encephalitis and poliomyelitis, cerebral sclerosis, cerebral tumor, disseminated sclerosis ("Intention tremor" (tremor of intended movements of the extremities, head, and tongue); nystagmus, disturbance of speech, forced laughing, spastic paresis, intercurrent apoplectic seizures). Little's disease (Intention tremor!). Hemorrhages of cerebrum and meninges, progressive paralysis. Meningitis (Especially at the upper extremities, may be caused by passive movements, as in paralysis agitans). Tabes infantilis, spinal syphilis, myelitis, and disseminating myeloencephalitis (Intention tremor!, visible disturbances, bladder disturbances), juvenile ataxia (Intention tremor, head tremor). Toxic neuritis (diphtheria, uræmia, lead, alcohol).

2. Functional neuroses: Hysteria, traumatic neurosis, exophthalmic goitre (Vibrating, especially hand and tongue); tetany; after epileptic paroxysm; chorea; myotonia of nurslings.

3. Grave acute infectious diseases: Typhoid and others.

129. FIBRILLARY CONTRACTIONS

Physiological at the protruded tongue.

1. Organic affections of the cerebrospinal system: Tumors, hemorrhages, encephalitis, sclerosis, meningitis, myelitis.

Paramyoclonus multiplex (Violent, paroxysmal, explosive twitchings of the muscles, especially in the dorsal position and in response to irritation; always beyond the eighth year).—Symptomatic in organic cerebral affections and functional neuroses (hysteria, neurasthenia, epilepsy).

2. Progressive muscular atrophy, neurospinal form (almost never myopathic form).

3. Grave acute infectious diseases.

130. CHOREIFORM CONDITIONS

Combined, involuntary muscular convulsions.

Simulated by epilepsy, petit mal.

1. Chorea minor infantum (Usually pale girls with neuropathic hereditary tendency, or boys, of 9 to 15 years; arrhythmic, not stereotyped, very multiform, often apparently affected movements in frequent application; often sudden onset, sometimes stronger on one side).—Polyarthritides, scarlet fever, influenza, diphtheria, typhoid, gonorrhœa, psychic trauma as causative factors. Diverse hereditary defects as constitutionally predisposing factors.

Chorea minor or paralytic chorea (Like the above, accompanying debilitated conditions and paralysis, trophoneurotic disturbances, also with compulsory ideas and maniacal impulses).—Chorea minor on hysteric soil?

Hemichorea minor.

2. Organic chronic (hemi) chorea (Usually together with or after spastic paresis; rigidity, increased reflexes, contracture and athetosis!) before and after hemiplegia, especially in infantile cerebral paralysis; hydrocephalus, tumors, sclerosis, arrest of development, inflammatory foci of the brain (especially at the capsule).—Progressive paralysis, idiocy.

3. Chorea major in hysteria and exophthalmic goitre (Usually stereotyped, rhythmic, dramatic movements executed with great intensity and excursion, free intervals!; only in older children; commencement generally sudden; psychic and somatic concomitant manifestations of a typical nature).

4. Electric chorea, Henoch-Bergeron's disease (Combined rhythmical irresistible movements executed with lightning-like rapidity, regularly recurring, lasting for years, sometimes inhibitable by compression of nerves; without signs of rheumatic or hysteric affection). Here may perhaps belong the "maladie des tics" with echolalia, coprolalia.

5. Hereditary progressive chorea (Huntington) (Hereditary family evil, occurring only in older children; always progressing for a considerable time, and fatal).

6. Paramyoclonus multiplex (Similar to electric chorea, but symmetrical, less extensive movements; only trunk and extremity muscles involved; not curable as the other).

[7. Choreiform unrest in juvenile ataxia (Friedreich) (Absence of reflexes, atactic character of movements), progressive paralysis of children, etc.]

131. ATHETOSIS

Slowly rounded off, peristaltic compulsory movements of the extremities, which continue during sleep and are confined to certain muscle regions.—Organic cerebral affections (especially of the internal capsule, often accompanying idiocy and epilepsy); hemiplegia, cerebral and meningeal apoplexy; cerebral sclerosis. Encephalomalacia after meningitis, cerebral tumor, cerebral syphilis; infantile cerebral paralysis; porencephaly, microcephaly.

132. ATAXIA

Simulated by chorea.

Up to a certain age is physiological for all movements.

1. *Cerebral Affections* (attacking especially the cortex, pons, cerebellum, or mesencephalon): Poli-encephalitis, tumor, sclerosis, hydrocephalus; progressive paralysis, congenital cerebellar aplasia.

2. *Spinal Affections:*

- (a) Friedreich's hereditary ataxia (Begins in the sixth to seventh year, lower—and upper—extremities; intention tremor, disturbance of speech, nystagmus, contractures: pes equinus with hyperextended toes, cerebellar walk, absence of patellar reflex; no visual disturbances, no bladder disturbances, no severe pains, no reflex immobility of pupil).
- (b) Infantile locomotor ataxia and posterior poliomyelitis. (Ataxia late symptom; visual disturbances, bladder disturbances, reflex immobility of pupil, Brach-Rhomberg's sign; muscular power retained).
- (c) Transverse and disseminated myelitis.

3. *Affections of the Peripheral Nerves and of the Muscles:*

- (a) Toxic polyneuritis (arsenic, alcohol, uræmia) and post-infectious neuritis (diphtheria, scarlet fever) (Lower extremities affected most; with debility or paralysis; patellar reflex absent; ocular muscles and sphincters free; RD; disturbance of sensation; usually favorable course).
- (b) Progressive amyotrophy (type Déjerine-Sottas) (Similar to tabes, but without disturbance of bladder and speech).

4. *Functional neuroses, hysteria.*

5. *General Pathological Conditions:* Cachexia, great debility after grave infectious diseases.

133. SYNDROME OF (PSEUDO) TABES

(Intention) tremor, vertigo, choreiform movements, symptoms of psychic defects, disturbance of speech.

1. Tabes infantum (+ progressive paralysis) (Visual disturbances, immovability of pupil, disturbance of bladder, epileptiform attacks, lancinating pains, anæsthesia, sphincter paralysis, ataxia occurring late or not at all).—Syphilis.

2. Friedreich's ataxia (Foot deformity, scoliosis, nystagmus, patellar reflex absent, no spasm; occurs in families).

3. Sydenham's chorea (chorea minor, especially mild chorea) (No distinct ataxia, usually rheumatic foundation).

4. Disseminated sclerosis and diffuse myelitis (Slight distinct ataxia; spasms, epileptiform seizures; patellar reflex usually retained).

5. Postinfectious pseudotabes, neuritic pseudotabes (RD.).

6. Cerebellar tumors (cerebral pressure!).

7. Hysteria.

134. HYPERTONIA OF THE MUSCLES

More or less persistent, paroxysmally increased.

Simulated by the "physiological myotony" of the newborn (tonic flexion from the articulations of ulna, hand, hip, and knee), and by sclerema adip. neonat., eclampsia.

I. UNIVERSAL

A. IN THE NEWBORN AND IN CHILDREN DURING THE FIRST TWO YEARS OF LIFE.

1. Trismus and tetanus neonatorum (Always dysphagia and lockjaw; mimic spasms; no tetanic trias, no Kernig's sign, often no fever).
2. Myotonia neonatorum (Permanent, painless flexion contractures of the extremities, especially permanent spasms in fingers and toes, usually developing gradually in the first few weeks of life; fist phenomenon spontaneously present or obtainable; carpopedal spasms; absence of the tetanic trias; no tension of fontanelles).—Cachexia in chronic septicæmia, gastro-intestinal derangements (athrepsia, dysthrepsia), hereditary syphilis, chronic dermatosis.

Pseudotetanus of the newborn (Persistent, tetanoid spasm with opisthotonos, but without trismus and pharyngeal spasms; always afebrile).—Higher degree of myotony.

3. Tetany (spasmophile diathesis) (Paroxysmal spasms without disturbance of consciousness, often nearly universal, but back of neck and masseters free; painful, with characteristic spastic positions, always the trias of latent tetany (phenomena of Erb, Chvostek, and Trousseau); mouth phenomena; often laryngospasm; only in artificially fed children from the second to the fourth year).—Status thymicus, rachitis, anæmia, disorders of nutrition, often hereditary tendency.
4. Organic cerebral affections (other cerebral manifestations: idiocy, reflex immobility of the pupil): Little's disease, especially in its special form of congenital, general rigidity; conditions consequent upon cerebral traumatism at birth, hemorrhages of the cerebrum and the meninges, acute encephalitis of nurslings, cerebral sclerosis, hydrocephalus chronicus, especially on syphilitic basis. Microcephaly, porencephaly, hypertrophy of the brain.
5. Meningitis, especially cerebrospinal meningitis (intermittent) (No participation of the mimic muscles, Kernig's sign positive, flexion spasm of the arms).

A. IN THE NEWBORN, etc.—Continued

6. Hydrocephaloid (Upper extremities in flexion or extension, rigid, pronated; thumb flexed into the palm).

[7. "Meningism" in acute infectious diseases, also miliary tuberculosis.]

B. OCCURRING ONLY IN MORE ADVANCED YEARS.

1. Traumatic tetanus.

2. Pseudotetanus of older children.

3. Organic cerebral and spinal affections: Syphilis, cerebral tumor, sclerosis, Pott's disease.

4. Meningitis.

5. Hysteria, catalepsy.

[6. Strychnine poisoning (Usually delirium, always mydriasis, no trismus).]

[7. Congenital myotonia (Thomsen's disease) (Family occurrence; tension, stiffness of the entire voluntary musculature, especially the extensors, with hypertrophy, normal nervous excitability, increased muscular excitability, no pains; slowly progressive; disturbance especially striking after muscular rest).]

II. LOCALIZED

1. Spastic paralysis (*see No. 165*).

2. Isolated carpopedal spasms (claw position of hand and foot, dactylotonus of arm and leg), tetany, arthrogryposis (trias of latent tetany, few pains, sometimes œdema, favorable course in a few days to a few weeks; simultaneously with digestive disorders).

135. KERNIG'S SIGN

The knee-joint of the patient while sitting (or after passive flexion of the thigh in the recumbent position) can be extended only out to about 135° (rigidity of the flexors!). Not significant if there are contractures; of no value in infants.

Cerebrospinal meningitis, suppurative meningitis (Frequent, but inconstant!).

Tuberculous meningitis (rarer and less distinct; disappears in the paralytic period; in infants mostly absent).

"Meningism" in acute infections, especially typhoid and other infectious intestinal conditions at early age.

136. TONIC-CLONIC SPASTIC CONDITIONS IN SINGLE GROUPS OF MUSCLES

(beside their participation in general convulsions)

1. *Trismus*. Simulated by sclerema adiposum and sclerœdema, by myotonia of athreptics.

2. *Rigidity of Neck and Opisthotonos.* Simulated by compulsory backward position of the head in cretinism, myxœdema, laryngeal stenosis, cervical spondylitis, vertebral polyarthritis, vertebral polymyositis, rheumatic polymyositis (trapezius, splenius, etc.), and other painful conditions of the neck.

3. *Spasms of the Extremities:*

- (a) Meningitis and meningeal hæmorrhage, chiefly tuberculous and cerebrospinal meningitis; also acute and chronic spinal leptomeningitis and pachymeningitis, hæmorrhages after traumatism at birth, spinal tumors.
- (b) Disturbances of circulation and organic changes in brain and spinal cord.—Hyperæmia, anæmia, hydrocephaloid, sinus thrombosis, sclerosis, encephalitis, tumors, hæmorrhages, hydrocephalus, transverse myelitis, Pott's disease.
- (c) "Meningism" in acute infections fulminating (scarlet fever, typhoid, cerebral pneumonia, influenza, miliary tuberculosis); in ectogenous intoxications (opium, meat and fungus poisons), and from reflex irritation (suppurative otitis media, etc.).
- (d) Functional neuroses: Tetany, myotonia, hysteria, epilepsy, eclampsia.
- (e) Traumatic tetanus and tetanus neonatorum (Often without fever; increased reflex excitability, absence of all disturbances of sensation). [Strychnine poisoning (Short duration, free intervals, mydriasis, severe involvement of the extremities). Rabies (Spasms of deglutition predominant, mental disturbances).]
- (f) In addition to trismus in grave inflammatory processes of the buccal and pharyngeal mucous membrane, tonsillar abscesses, parotitis [trichinosis].
- (g) Rigidity of the neck in occipital neuralgia.

137. SPASMS OF THE SPINAL ACCESSORY NERVE

1. *Organic Cerebral Affections*, especially if the cerebellum is involved: Encephalitis, sclerosis, chronic hydrocephalus (often along with idiocy and organic chorea), meningitis.

2. *Functional Neuroses:* Eclampsia, epilepsy, chorea, hysteria, convulsive tic of the muscles of the neck.

3. "*Spasmus Nutans*" (Effortless, stereotyped, pagoda-like rocking, turning, nodding, shaking of the head, often with nystagmus; non-conjugated, immobile look, stupor; generally ceasing during sleep and when eyes are closed). Simulated by masturbation, movements because of itching, and as an expression of well-being.

3. "*Spasmus Nutans*," etc.—*Continued*

- (a) In the first 3 years of life: (Congenital) ocular affections, primary nystagmus; general debility, rachitis.
- (b) Beyond the fourth year of life: Organic cerebral affections, hysteria (Here without nystagmus).

138. LARYNGOSPASM

See No. 68, II., A. 2.

True tetany of rachitics (Facial and Trousseau phenomena).

Laryngeal affections in spasmophilic diathesis. [As a reflex phenomenon in tonsillar hypertrophy, tumors of the mediastinal glands?] [Genuine epilepsy?]

Cerebral processes, like serous meningitis, chronic hydrocephalus, [hypertrophia, cerebral tumor, and other organic cerebral affections].

139. SPASMS OF THE DIAPHRAGM

Clonic spasms, see *Singultus*. In tonic spasms, epigastrium bulging, forced respiration at the upper thorax; cyanosis, dyspnoea, dread of suffocation; deep pulmonary borders.—Cervical Pott's disease, tetanus, tetany, epilepsy, hysteria. [Rabies, strychnine poisoning.]

140. SPASMS OF THE FACIAL NERVE

Often with spasm of the eyelid, blepharospasm.

Cerebral conditions: Irritations of the cervical cortex (central convulsions), basal, pontine affections, meningitis, infantile cerebral paralysis (post-hemiplegic).

Functional neuroses: Eclampsia, tic convulsif, maladie des tics. Peripheral nervous diseases: Neuritis of trigeminal nerve.

141. SPASMS OF DEGLUTITION

[Rabies,] tetanus, irritation of the trigeminus, hysteria (globus).

142. GENERAL REFLEX CONVULSIONS

Rapid jerking and violent inspiration on percussion of the body and on other irritations.—Physiological in the newborn and in young infants. Meningitis (especially suppurative), traumatic tetanus. pseudotetanus, general neuropathy [rabies, strychnine poisoning].

143. GENERAL CONVULSIONS WITH LOSS OF CONSCIOUSNESS

Apoplectiform and epileptiform attacks.

Simulated by fainting, tic, chorea.

I. ORGANIC CAUSES, ACCOMPANYING CEREBROSPINAL LESIONS

Always together with other cerebrospinal signs; convulsions never occur first! Convulsions, often unilateral, of long duration, severe paral-

ysis, stupor, hypertonia, myosis, pulse anomalies may or may not be accompanied by fever. Not infrequently neuroretinitis with choked disk.

A. IN INFLAMMATORY PROCESSES WITH ACUTE ONSET AND ACCOMPANIED BY regular and considerable FEVER*.

1. Acute meningitis (Vomiting, headache, rigidity of the neck, constipation, anomalies of pulse and respiration, neuroretinitis). [Tuberculous meningitis, atypical commencement.]
2. Acute encephalitis, cerebral abscess (Choked disk; usually paralysis, contractures, sensory and psychic disturbances).
3. Septic sinus thrombosis (Spasms occurring with chills, predominantly tonic, interrupted by short clonic spasms; ocular muscles much involved; tachypnoea).—Gastroenteritis, sepsis, pneumonia, measles, tuberculosis.

B. IN PROCESSES WITH ACUTE ONSET, BUT USUALLY AFEBRILE OR SUBFEBRILE.

1. *Embolism, Cerebral Thrombosis* (Existing cardiac affection, usually tachycardia; after the attack often rise in temperature).
2. *Hæmorrhages of the Brain and Meninges* (Face hyperæmic, temperature subnormal, bradycardia, conjugate deviation):
 - (a) In the newborn after traumatism at birth. Syphilitic arteritis.
 - (b) In older children: spastic cough attacks; congenital cardiac defects.
3. *Hydrocephaloid*.
4. *Cerebral Hyperæmia*.—Marantic sinus thrombosis, spastic cough, cardiac defects.
5. *Internal Cephalæmatoma*.

C. IN AFEBRILE PROCESSES WITH SLOW ONSET OR EXISTING FROM BIRTH.

1. Chronic internal hydrocephalus (frequently together with tetany).
2. Microcephaly, porencephaly, cerebral hypertrophy (Congenital, with permanent spasms or spastic paralysis and idiocy; often athetosis and bulbar symptoms).
3. Cerebral and spinal tumors (signs of cerebral pressure: bradycardia, vomiting, vertigo, choked disk; convulsions often limited to single groups of muscles, at first without disturbance of consciousness. Convulsions frequently recurring, no initial cry; paralysis).—Especially cortical tumors. Tuberculosis, syphilis.

*Fever may exceptionally be absent. (Tubercular meningitis, cerebral abscess.)

C. IN AFEBRILE PROCESSES, etc.—*Continued*

4. Jacksonian epilepsy (Spasm commences in definite motor areas, remains at first without disturbing consciousness; other cerebral signs!).—Various lesions of the cervical cortex.
5. Diffuse cerebral sclerosis (Setting in gradually, with spastic paralysis, oculopupillary signs).—Generally syphilitic arteritis.
6. Disseminated cerebral sclerosis.
- [7. Incipient progressive paralysis (Vertigo, headache, paralysis of the ocular muscles; disturbance of consciousness occurring first, then the attack; in the interval, disturbances of vision and intelligence).—Syphilis.

II. NO ORGANIC CAUSES, UNACCOMPANIED BY CEREBROSPINAL AFFECTION

A. "IDIOPATHIC," "ESSENTIAL" (Existing independently, or occurring as a sign of habitual overexcitability; always recurring again for months and years, taking an afebrile course).

1. Genuine epilepsy (Often in the beginning—and sometimes throughout—attacks occur only at night, aura, initial cry, complete loss of consciousness, for a certain time and always in the beginning of the attack; its total duration ten minutes at the most; often subnormal temperature, biting of the tongue, sudden fall and injury, involuntary evacuations. Reflex immobility of pupil, stuporous expression and pallor of the face. After the attack, sleep, albuminuria, no paralysis, almost never laryngeal spasm or apnœa. Loss of memory and change of character, otherwise intervals clear. No cerebral pressure, no tetany triad; outside of hereditary tendency, no cause can usually be ascertained; occurrence generally not before the end of the second year).
2. Hysteria (Form of attack dramatic, spastic movements co-ordinate, with wide excursions. No reflex immobility of pupil, only mydriasis; no crying out, no sudden fall, no complete absence of consciousness, seldom tongue-biting, and involuntary evacuations; after the attack no stupor. Facial expression affectionate; attacks occur only during the day and are often of long duration, stigmata in the intervals. Almost exclusively in children over 5 years old).
3. Genuine eclampsia (often along with tetany in rachitics) (Usually alternating with laryngospastic attacks, triad of latent tetany. Rachitis).
- [4. Eclampsia nutans, true salaam spasms (Folding the body

A. "IDIOPATHIC," etc.—*Continued*

forward in a jerk with short absence of consciousness; commencement often in the first year of life, later sometimes "transition into epilepsy").

B. SECONDARY. Unforseen spasms; symptomatic eclampsia, convulsions in "meningismus" (In children during the first 2 years of life, less frequently later on; in spasmophilic patients, occur in the wake of certain diseased conditions; in the beginning usually full consciousness and reflex immobility of the pupil).

1. Hæmatogenous in acute infectious diseases; especially in the beginning and in severe "cerebral," highly febrile course: Scarlet fever, measles, sepsis, pneumonia, influenza, non-specific angina; miliary tuberculosis. Also in whooping-cough. [Varicella, typhoid, mumps, anterior poliomyelitis (Without complete loss of consciousness and not recurring)].
2. Hæmatogenous in ectogenous intoxications: Alcohol (also effective through the body of the wet-nurse), phenol, bromoform, iodoform, carbon monoxide, santonin, alkaloids (opium); fungus poisons, aspidium filix-mas, chlorate of potash, lead (diachylon plaster!), arsenic.
3. Hæmatogenous in endogenous intoxications: Diabetic coma, cholæmia, puerperal eclampsia of the mother. Many acute infectious kinds of gastro-enteritis, dyspepsia with acetonæmia, constipation, helminthiasis (ascarides), suffocation and terminal CO₂ poisoning (*e.g.*, in bronchiolitis). Uræmia, tetanus, Addison's disease.
4. Neurogenous, reflex effect; violent sensory irritations starting from certain zones: (In younger and spasmophilic children the slightest causes, such as a cold in the head, a slight angina, etc.) Difficult dentition, intestinal parasites, colic, constipation, rectal and anal fissure, invagination, dysentery. Dysuria, urolithiasis with phimosis, cryptorchis. Skin burns, injuries, vaccination, other painful dermatoses. Nasal affections, adenoid vegetations, otitis, foreign bodies in the ear. Mental shock?).

144. TEETH GRINDING, INVOLUNTARY SUCKING AND CHEWING,
PICKING AT THE NOSE, TUGGING AT THE LIP
RUBBING THE EYES

In healthy children (while asleep) occasionally (itching and pain, local), but also often habitual for years; also other similar involuntary motions, such as the various forms of humming, wagging the head while asleep, etc.

"Meningismus," infections (scarlet fever, typhoid, erysipelas, etc.), toxic, and reflex (indigestion).

Meningitis (here also carphology).

Cerebrospinal disorders, disturbances of circulation, and organic changes, especially bulbar irritations: Cerebral anæmia, tumor, acute anterior poliomyelitis, microcephaly, progressive paralysis.

(Rubbing the occiput against the pillow habitually in rachitis; occasionally in otitis and itching eruptions.)

145. PAVOR NOCTURNUS

Attacks of fright at night without awaking, accompanied by screams of terror, dread, excitement, 2-3 hours after going to sleep.

Here belong too, crying out at night, twitching; deglutition spasms during sleep; somnambulism.

A. HABITUAL (Frequently recurring for months and years. Usually the joint action of a constitutional and an exciting factor (1 and 2, below.

1. Neuropathic hereditary tendency, degeneration, hysteria (early symptom). Anæmia, "neuro-arthritis" (together with epilepsy equivalent?), masturbation, chronic vulvo-vaginitis.

2. Constipation, dyspepsia, intestinal parasites.

Abuse of alcoholic liquors.

Adenoid vegetations, hypertrophy of faucial tonsils, nasal stenosis, cardiac affection (attacks of suffocation!).

All kinds of abuses, or mistakes in nursing, attendance, and education of the child.

Fever; heavy dreams.

B. OCCASIONAL.

Cerebral tumor, tuberculous meningitis (precursor!). Chorea. Otitis.

Atropine poisoning.

Whooping-cough.

146. INSOMNIA

Daily total of sleep of nurslings: 20 to 16 hours.

Daily total of sleep in infancy: 16 to 12 hours.

Daily total of sleep in childhood: 11 to 9 hours.

A. IN NURSLINGS.

1. Pains and itching.—Colic, dyspepsia from overfeeding, otitis, intertrigo, eczema, lichen strophulus, furunculosis.

2. Insufficient aeration of the lungs.—Rachitis, all stenoses of the upper air tracts, including simple coryza, deficient ventilation (and overheating) of the bedroom.

B. IN OLDER CHILDREN.

1. General neuropathies and constitutional disorders; see the conditions which lead to pavor nocturnus (*No.* 145, *A*).
2. Conditions of cerebral irritation, hyperæmia, especially in acute infections (*e.g.*, in typhoid). Progressive paralysis.
3. Tetany, chorea minor.
4. Uræmic "meningismus." Diabetes mellitus.

147. NERVOUS STATE, NEUROPATHIA

Impatience, ill-temper, bad sleep, headache, lassitude, twitching, tremor, pavor nocturnus.

Chronic dyspepsia and abdominal dilatation, as consequences of improper dietary regime.

Anæmia, "neuro-arthritis" (often with orthostatic albuminuria).

Status lymphaticus, hysteria, exophthalmic goitre, epilepsy.

Chronic endocarditis.

Habitual masturbation.

Parasyphilis.

Local affections with reflex effect, intestinal parasites, vulvo-vaginitis.

Prodrome of tuberculous meningitis.

148. WEAK-MINDEDNESS, IMBECILITY, DEMENTIA, IDIOCY

Early symptoms: Awkwardness in sucking, inability to grasp, lack of interest, inability to learn to speak and walk.

Simulated by dulness of hearing, micromelia.

1. Organic cerebral affections.—Hydrocephalus, microcephalus, porencephalus, premature ossification, cerebral hypertrophy, cerebral hernia.

Embolism and hæmorrhage of the brain and meninges (whooping-cough, congenital cyanosis,) encephalitis, sclerosis, tumor, infantile cerebral paralysis; all these conditions especially if they are engrafted on a syphilitic basis.

Post-meningitic defects (especially after cerebrospinal meningitis).

Progressive paralysis (Epileptiform seizures, syphilis).

Hereditary ataxia, second stage.

2. Homologous heredity; "degeneration," hereditary defects from alcoholism, consanguinity (?), etc.

[Hereditary amaurotic idiocy (In Jewish families, complete amaurosis, nystagmus, strabismus, flaccid or spastic paralysis of the muscles of the back and extremities).]

[Hereditary syphilitic idiocy (Relatively progressive mental backwardness, which is often noticed only later; infantilism, oddities; usually along with other cerebral symptoms).]

3. Myxœdema, cretinism, mongoloid (*see Table. belonging to No. 8*).

4. Nasal and pharyngeal aprosexia (chronic rhinitis, adenoid vegetations) (Weakness of memory, mental lassitude, backward intelligence, appearance and bodily signs).

149. STUPEFACTION, APATHY, SOMNOLENCE, COMA, STUPOR

A. "Meningismus."

1. Acute infectious diseases (beginning and course).—Typhoid, influenza, severe diphtheria, croupous pneumonia, whooping-cough, dysentery, erysipelas, Weil's disease, acute poli-encephalitis.

2. Acute ectogenous intoxications.—Phenol, antipyrin, quinine, morphine, fungoid poison, chloral hydrate, alcohol, and other narcotics, illuminating gas, and marsh gas.

3. Endogenous intoxications.—Carbonic acid poisoning. Uræmia, (Edema, anuria, vomiting, frequently convulsions, Cheyne-Stokes). Diabetes mellitus (Hypothermia, seldom convulsions, deep respiration, acetone odor). Cholæmia. Dyspepsia.

4. On "reflex" basis: Intestinal parasites, masturbation.

B. Disturbances of circulation in the cerebral area.—Hydrocephaloid, concussion, sinus thrombosis, heat stroke.

C. Organic disorders of the cerebrospinal system.

1. Tuberculous, suppurative, epidemic, and serous meningitis.

2. Embolism, hæmorrhages of the brain (especially also in the newborn).

3. Encephalitis, cerebral tumor (weakness of the memory, "hibernation"), cerebral abscess, cerebral hypertrophy, progressive paralysis.

D. Extreme cachexia, agony, grave forms of idiocy (*see No. 148*), pernicious anæmia, in the third stage.

150. TENDENCY TO FAINTING. ABSENT-MINDEDNESS

1. Anæmic conditions: Simple and pernicious anæmia, chlorosis, anæmia after exsanguination and in chronic dyspepsia.

2. Conditions of cardiac weakness: Congenital heart disease, myocarditis, cardiac debility in acute infectious diseases, typhoid, miliary tuberculosis, and others.

3. Hypertrophy of the faucial and pharyngeal tonsils.

4. Chronic intoxications.

5. Epileptic petit mal (Albuminuria after the attack).

151. IRRITATIVE MENTAL DISTURBANCES, OCCURRING SUDDENLY

Mania, raving, excitement like intoxication.

1. In and after acute infectious diseases: Typhoid, scarlet fever, whooping-cough, measles, mumps, influenza.

2. Pyæmia and septicæmia; *e.g.*, suppurative peritonitis.

3. Ectogenous intoxications: Iodoform, codeine, cocaine, alcohol, salicylic acid, santonin, quinine, phosphorus; botulism.

4. Chorea minor, epilepsy.

152. DEPRESSIVE MENTAL CHANGES OCCURRING GRADUALLY

(aside from psychoses)

Ill-humor, irritability, weeping, distractedness, forgetfulness; easily fatigued, rapid change of disposition.

1. Meningitis: Important prodrome of tuberculous meningitis! at intervals in cerebrospinal meningitis.

2. Other cerebral affections: Tumor, incipient paralysis, Little's disease.

3. Functional neuroses and general neuropathy (*see No. 147*), especially incipient chorea minor.

4. Habitual anæmia: "School-sickness," splenic and simple anæmia, chlorosis.

5. Sequelæ of acute infectious diseases; *e.g.*, convalescence from typhoid.

6. Diabetes mellitus.

153. INCREASED MECHANICAL EXCITABILITY

A. OF THE NERVES.

Chrostek's Facial Phenomenon.—Exceptionally in older children without any other sign of illness, frequently in the newborn and in nurslings.

1. Tetany (of rachitics, in the first 2 years of life fully developed, later persisting in a few signs).

2. Cerebral and meningeal affections, especially Little's disease.

3. Postinfectious neuritis (typhoid and diphtheria).

4. Myxædema, mongoloid.

5. Anæmia.

Trousseau's Phenomenon, Mouth and Lip Phenomena (Claw position upon pressure in the internal bicipital groove or upon grasping the arm; contraction of the orbicularis oris muscle upon percussion, isolateral or contralateral).

6. The latter frequently in healthy children of the first year of life.

7. Tetany, spasmophilic diathesis.—In emaciated children with

A. OF THE NERVES.—*Continued*

intestinal affection on the one hand, and in overfed, fat, doughy, constipated, anæmic children on the other.

B. OF THE MUSCLES.

Increased Prominence at the Muscles.—Cerebral disorders (e.g., poliomyelitis); congenital myotonia of Thomsen (Myotonic reaction), tetany, cachexia, emaciation.

154. CHANGES IN THE ELECTRICAL EXCITABILITY OF NERVES AND MUSCLES

The electrical excitability of the median nerve in health when tested properly with an Erb's electrode, applied at the motor point of the nerve, is as follows:

	Farradic.	Galvanic: CCc	ACc	AOc	COc
Children over 8 weeks of age, about 110 mm. R.A.		1.4,	2.2,	3.6,	8.2, M.A.
Children under 8 weeks of age, about 83 mm. R.A.		2.6,	2.9,	5.1,	9.3, M.A.

1. *Reaction of Degeneration:*

Occurs almost invariably in diseases of the peripheral neurons.

- (a) Spinal affections. Acute and chronic infantile paralysis, transverse lumbar myelitis, amyotrophic lateral sclerosis, bulbar paralysis, progressive muscular atrophy (neurospinal type).
- (b) Chronic neuritis, of toxic, rheumatic, or infectious origin with the exception of postdiphtheritic neuritis, injuries of the peripheral nerves.

2. *Lessened Irritability without Reaction of Degeneration:*

- (a) Spinal affections. Certain lesions of the pyramidal tract, lateral sclerosis, bulbar paralysis, transverse lumbar myelitis.
- (b) Peripheral diseases of the nerves and muscles, neuritis, including postdiphtheritic paralyses, all forms of muscle dystrophies, congenital myotonia.
- (c) Severe anæmias.

3. *Increased Excitability:*

- (a) Tetany (Especially galvanic! AOc > ACc, COc under 5 milliamperes). Progressive spinal muscular atrophy, congenital myotonia of Thomsen (Only the muscles, not the nerves).
- (b) Cerebral disorders: Poliomyelitis, tumor, status post-hemiplegicus, fresh cerebral paralysis.
- (c) Chorea minor.
- (d) Some forms of anæmia.

155. CHANGES IN THE TENDON REFLEXES

Increased patellar reflex and ankle clonus.

NOTE: In healthy children in the first year of life the patellar reflex is stronger than in adults.

1. In nearly all spinal and cerebral spastic paralyses of the lower extremities (*see No. 165*); also in progressive paralysis. In transverse myelitis, if the focus is situated above the 2nd or 3rd lumbar vertebra.

2. Various forms of meningitis before the stage of paralysis.

3. General hypertonia of the muscles (*see No. 134*).

4. Functional neuroses. Hysteria, tetany (chorea minor?).

5. Chronic nephritis, especially contracted kidney (uræmia?).

Diminished or absent patellar reflex (Westphal's sign).

1. In nearly all flaccid paralyses of spinal and peripheral origin and in the early stages of some forms of spastic paralyses. In transverse myelitis of the lumbar region when the focus is at or below the 2nd or 3rd lumbar vertebra.

Also in hereditary ataxia, progressive paralysis, infantile tabes; before and after apoplectic and epileptic attacks.

2. Conditions of flaccidity and atrophy of the muscles without real paralysis. Chorea mollis, myopathic form of progressive muscular atrophy, third stage (if quadriceps is involved), masturbation [congenital myotonia, diabetes mellitus].

3. "Meningismus" in acute infectious diseases and intoxications with a "cerebral course," especially croupous cerebral pneumonia (early symptom!), often already a long time before occurrence of physical signs.

4. At the end and in the gravest forms of cachexia.

NOTE: After many cases of illness, as for instance postdiphtherial neuritis, Westphal's sign remains for a long time and may then mislead as to intercurrent affections to which it is referred.

156. ANOMALIES OF SKIN REFLEXES

1. *Increase of the Skin Reflexes*.—Is general in and after spastic paralysis, in hypertonia.

2. *Diminution or Absence of the Skin Reflexes**.—Is general in nearly all flaccid paralyses, including progressive muscular atrophy, hereditary ataxia, masturbation (?), hysteria (very frequently absence of plantar reflex; also of the soft palate and corneal reflex), meningitis III.

3. *Babinski Reflex* (Dorsal flexion of the great toe in all joints, and sympathetic simultaneous movement of the other toes (in a less degree) on gently stroking the sole of the foot near the inner border):

(a) Normal in children up to the third year of life; may occur later in rachitics and during sleep.

*Cremaster reflex frequently absent in healthy children.

3. *Babinski Reflex, etc.—Continued*

- (b) After the fourth year of age and when awake: meningitis, chronic hydrocephalus, cerebral and spinal spastic paralysis, (apoplexy, spastic spinal paralysis). [Hysteria.]

157. PARESIS AND PARALYSIS OF THE VAGUS NERVE

Nausea, vomiting, abdominal pain, tachycardia, arrhythmia, intermittent small pulse, palpitation, precordial fright, dyspnoea, cyanosis, pallor and cold extremities, prostration, perhaps sudden death.

1. Partial manifestation of bulbar paralysis (*see No. 158*).
2. Neuritis of the vagus nerve: Toxic (atropine, digitalis), infectious (diphtheria).
3. Lesion of the vagus through mediastinal processes.
4. Reflex paralysis on violent sensitive peripheral irritations.

158. MULTIPLE PARESIS AND PARALYSIS OF CEREBRAL NERVES WITH MEDULLARY NUCLEUS

("bulbar and pseudobulbar paralysis")

Affected: Nuclei of the hypoglossus, facial, glossopharyngeal, spinal accessory [trigeminus, oculomotor, and abducens nerves].

Dyskinesia of the tongue, lips, palate, larynx. Atrophy of the affected nerves or muscles.

Disturbance of articulation (lingual sounds in "parallelogram," labial sounds in "public symphony"), lingual atrophy with fibrillar jerks, dysphagia; facial diplegia, salivation, disturbance of mastication, faulty deglutition; tachycardia, tachypnoea, cyanosis, jactitation; oculo-pupillary signs [myosis, conjugate deviation].

1. Acute infectious and toxic conditions, especially with hyperpyretic course: Scarlet fever, measles, botulism.

2. Organic lesions, especially ascending spinal processes: Amyotrophic lateral sclerosis III, Landry's paralysis, progressive spinal muscular atrophy, diffuse cervical myelitis, Pott's disease, acute anterior poliomyelitis, acute inferior policephalitis, multiple sclerosis, hereditary ataxia, tumors, hæmorrhages of the medulla, basilar and spinal meningitis III. Cortical lesions. Thrombosis of the transverse sinus. Microcephalus and pseudomicrocephalus.

[3. Acute and chronic progressive bulbar paralysis.]

159. PARESIS AND PARALYSIS OF THE SOFT PALATE

Dysphagia and regurgitation, nasal voice, unilateral or bilateral immobility, drawling speech.

Simulated by tonsillar hypertrophy, other pharyngeal and nasal affections, especially stenosis.

As partial manifestation of bulbar paralyse (*see No. 158*).

1. "Inflammatory mechanical paralysis" in grave local processes, angina and pharyngitis with infiltration and rigidity.

2. Genuine paralysis.—Usually toxic (botulism) or postinfectious (diphtheritic) neuritis.

[Cerebrospinal and meningeal affections: Poli-encephalitis, poliomyelitis, meningitis.]

[3. As partial manifestation of a paralysis of the facial nerve.]

160. PARESIS AND PARALYSIS OF THE HYPOGLOSSAL NERVE

Tongue protruded toward the affected side; disturbance of speech (lingual sounds in "Störing").

1. *Central Paralysis* (Electrical excitability maintained, no atrophy of the lingual muscles; sometimes bilateral):

(a) Cortical (Often + paralysis of facial nerve + brachial monoplegia).

(b) Capsular and peduncular (Crossed hemiplegia, or + oculomotor and facial paralysis).

(c) Lesion of the central convolutions, embolism and apoplexy, sclerosis, encephalitis, tumor.

2. *Nuclear Paralysis* (Together with paralysis of the IX, X, XI, XII cranial nerves; dysphagia, aphonia, dyspnoea, changes of the pulse).—Part manifestation of bulbar paralysis (*q.v.*). Infantile karyolysis; nuclear aplasia.

3. *Peripheral Paralysis*: Trunk or branches (Absence of bulbar and cerebral signs, RD., atrophy, wrinkling and furrowing of the tongue, and tremor; almost exclusively unilateral).—Trauma, cicatrices, glandular tumors, affections of the cervical vertebrae, neuritis.

161. PARESIS AND PARALYSIS OF THE FACIAL NERVE

Palpebral fissure open, weeping (in spite of diminished secretion), forehead smooth, mouth angles deep, nasolabial fold obliterated, face semi-expressionless; difficult talking, blowing, whistling, laughing, showing of teeth; lip, chin, tip of nose, tongue [and uvula] drawn toward the healthy side. [Soft palate hanging down, salivary secretion diminished, hearing and taste disturbed.]

Simulated by spasm of the facial nerve on the other side, amyotrophic muscular paralysis (Landouzy type), congenital defect of the nerve.

A. *CENTRAL PARALYSIS* (Middle and lower branch more strongly affected, mimic paralysis absent or indistinct, reflex excitability of the paralyzed parts maintained, reflex movements of expression present, also electrical excitability, no RD. Other cerebral manifestations).

1. Organic changes, chronic hydrocephalus, embolism and thrombosis, hæmorrhages, sclerosis, tumors, encephalitis,

A. CENTRAL PARALYSIS, etc.—Continued

cerebral syphilis*, cortical defects after trauma at birth*.

2. Acute infectious diseases, cerebral pneumonia, sepsis neonatorum*, acute anterior poliomyelitis.

B. NUCLEAR PARALYSIS.

Infantile karyolysis* and nuclear aplasia* (along with paralysis of the ocular muscles), bulbar paralysis, Landry's paralysis III, poliomyelitis.

C. PERIPHERAL PARALYSIS (Frequently complete paralysis, affecting simultaneously all three branches; true mimic paralysis; extinction of the movements of expression and the reflexes of the affected parts, RD. and atrophy present or to be expected, if the cause persists; no cerebral manifestations).

1. Basal lesion (All branches affected, other cerebral nerves also involved, especially VI and VIII).
2. Lesion of the Fallopian canal and the internal auditory meatus (Involvement of the eighth cerebral nerve; disturbances of taste, hearing, movement of soft palate, salivary secretion only partial; inner branches partly spared.)
3. Lesion outside the stylomastoid foramen (Function of all inner branches intact).
4. Lesion peripherally from the ramification (Function of single principal branches intact).

I. Basal meningeal affections: Meningitis* of all forms (early symptom of tuberculous meningitis!).

II. Infectious, toxic, and rheumatic neuritis.—Scarlet fever, typhoid, diphtheria, whooping-cough.

III. Swellings, inflammatory and suppurative processes in the neighborhood of the nerves, tumors, cicatricial pressure.—Ulceration of the mastoid process; caries of the petrous bone, severe rachitic changes of the cranial skeleton*, affections of the cervical tumors, otitis media, mumps with suppuration. Operative severing of the nerves.

IV. Traumatism, especially at birth*, forceps lesions, and pressure in the uterus*.

V. Functional neuroses: Chorea, epilepsy, hysteria.

162. PARESIS AND PARALYSIS OF THE NECK

Postdiphtheritic neuritis. Acute and chronic anterior poliomyelitis, transverse cervical myelitis.

Spinal progressive muscular atrophy of early infancy.

Amaurotic and microcephalic idiocy.

*The causes marked * are especially referable to infants.

163. PARESIS AND PARALYSIS OF THE RESPIRATORY MUSCLES

A. DIAPHRAGMATIC PARALYSIS (Sinking in of the abdomen, rising of the liver at forced inspiration, bulging out of the abdomen and descent of the liver at expiration, elevation of lower borders of the lung, difficult coughing and vomiting).

1. Spinal lesions: Transverse cervical myelitis, infantile tabes, Landry's paralysis.
2. Affection of the phrenic nerve: Neuritis, toxic, infectious (diphtheria), and rheumatic. Lesion of the nerve in the neck through changes of the neighboring organs.
3. Hysteria.

B. PARALYSIS OF THE (ABDOMINAL) EXPIRATORY MUSCLES (Powerless cough, difficult speech and defecation. Inability to raise the body from the dorsal position, to turn the trunk, etc. Distention of the abdomen on pressure and crying. Flaccidity on palpitation. Absence of reflexes).—Acute anterior poliomyelitis (possibly unilateral, often partial paralysis, recti muscles free), transverse myelitis. Dorsal meningomyelocoele and other spinal lesions in the dorsal area (often unilateral). Postdiphtheritic paralysis.

164. FLACCID PARESIS AND PARALYSIS OF THE EXTREMITIES *

Nearly always RD. or diminished electrical excitability. Diminution of the deep (and superficial) reflexes, degenerative muscular atrophy; sometimes short, spastic initial stage.

Simulated by the conditions mentioned in No. 166.

A. SPINAL.

1. *Acute Anterior Poliomyelitis* [seldom taking a chronic course] (Usually sudden onset with alarming manifestations—fever, vomiting, seldom convulsions; paralysis at first very extensive, tetraplegia, triplegia, paraplegia, increases further in intensity, decreases in extent, remains limited to a preferred, synergetically coördinated group of muscles; early atrophy of the muscles and the entire supporting apparatus in the affected region. Skin livid, cool. Almost never disturbance of sensibility, sphincters, or cerebral nerves. Disappearance of the deep and superficial reflexes).—Idiopathic or after acute infectious diseases.
2. *Transverse Myelitis*, focal affection of the substance of the spinal cord, attacking a large part of a transverse section. Focus in the segment of the region affected by the paralysis. (Onset varies according to the nature of the disease; always disturbance of sphincters, frequently backache):

*Paralysis of single spinal nerves analogous to that of adults.

A. SPINAL.—*Continued*

- (a) Inferior cervical muscles (Flaccid paralysis of both upper extremities, together with spastic paraplegia, oculopupillary signs,—ptosis, myosis,—with paralysis of the trunk muscles; incontinence with over-filled bladder).—Pott's disease, hæmorrhage, tumors (tuberculosis, syphilis, glioma), traumatism at birth and otherwise; inflammatory processes following acute infectious diseases (measles, whooping-cough, typhoid).
- (b) Lumbar muscles (Flaccid paraplegia; atrophy and RD. may be absent; disturbance of sensibility in the pubic region and in the lower extremities, decubitus; incontinence with moderately filled bladder).—Occurrence as under 2, (a). Here also belong spinal myelocoele, spina bifida.
3. *Landry's Paralysis* (Sudden, sometimes with manifestations of an acute infection: paraplegia setting in with pain and fever and rapidly ascending and progressing to trunk, arms, and bulbus. Seldom RD.; no atrophy; sensibility and sphincters intact).—After diphtheria, influenza, typhoid, gonorrhœa.
- [4. *Syringomyelia*.]
5. (*Lepto*-) *Meningitis Spinalis* of various nature (Diplegia developing under the meningeal picture, fever and back-ache; at first often spastic (also contraction of back and neck!), later flaccid; at first very pronounced hyperæsthesia, later very pronounced anæsthesia, disturbance of sphincters, decubitus).—As part lesion in cerebral meningitis, also in spinal meningitis accompanying traumatic and acute infections: Trauma of birth, vertebral fracture, vertebral affection, pleuritis, sepsis, pneumonia, typhoid.
6. *Spinoneural, Progressive Muscular Atrophy* (Insidious, commencement at the pelvic girdle or lower extremities, later extension; always symmetrical, atrophy and paralysis always parallel; disturbance of sensibility occurs, pseudotabes; sphincters intact, frequently fibrillary twitching. Occurs in families!).

B. PERIPHERAL.

1. *Multiple Neuritis* (Beginning in some cases quite suddenly (infectious form), in others more insidiously (toxic form); progress mostly bilateral, symmetrical; paralysis of the distal ends of the extremities. Progressing centripetally, complete repair possible even after months. Disturbance

B. PERIPHERAL.—Continued

of sensibility, especially pain of nerve pressure, very pronounced, always preceding the paralysis, seldom sphincteric disturbance, no decubitus, sometimes ataxia).—Infections: Diphtheria, influenza, typhoid, scarlet fever, measles, whooping-cough, mumps, dysentery, sepsis, polyarthritis, pneumonia, tuberculosis, [syphilis*]. Intoxications: Alcohol, carbonic monoxide, other vegetable poisons; lead, mercury, arsenic, phosphorus.

Postdiphtheritic neuritis (Beginning 2–3 weeks after the local affection; after preceding disturbance of the soft palate† and ocular muscles, weakness of the lower extremities, then the upper extremities. Symmetrical extension, favorable tendency to heal; rapid convalescence. RD. and atrophy may be entirely absent).

2. Nerve Trauma during Birth:

(a) Paralysis of the superior cervical plexus (Flaccid paralysis of an arm, noticed soon after birth, hanging down in pronation, extension, involution; muscles especially affected are infraspinatus, deltoid, biceps, brachialis anticus, supinator longus; fingers can be moved; spontaneously favorable course; generally no atrophy, disturbance of sensibility absent or slight, no RD.; sometimes accompanied by luxation or fracture).—Birth trauma: pressure, pull at arm, deep forceps.

(b) Paralysis of the inferior cervical plexus; dorsal nerves and sympathetic fibres often involved: Klumpke's paralysis (Noticed after birth, forearm and hand especially involved, including small muscles of the hand (and ocular muscles: unilateral ptosis and myosis); anæsthesia of forearm).—Etiology like 2,(a).

[(c) Paralysis of the crural plexus.]

3. Painful Brachial Paralysis (Sudden paralysis of one arm, hanging down; hand pronated; may be chiefly "pain paralysis"; cure always spontaneous, rapid, uneventful).—Trauma (pull, tear).

C. IN FUNCTIONAL NEUROSES: Hysteria, traumatic neurosis, exophthalmic goitre (Paralysis often setting in suddenly, functionally arranged, paralytic, monoplegic, hemiplegic; more pronounced at the proximal parts of the extremities; rapidly changing; no RD., usually no atrophy; no decubitus, no sphincteric paralysis; often characteristic mental changes).

*Syphilitic neuritis is said to occur in newborn and under the picture of Klumpke's paralysis.
†In extra-oral diphtheria, also other and corresponding localization (finger, umbilicus).

165. SPASTIC PARESIS AND PARALYSIS OF THE EXTREMITIES

Muscles almost always tense, (superficial) and deep reflexes increased; often manifestations of motor irritation; atrophy setting in only slowly, usually not considerable; no RD.; at the very beginning paralysis sometimes flaccid.

Simulated by conditions of general or local hypertonia of the muscles (*see No. 134*).

A. MENINGEAL PROCESSES: Cerebrospinal, tuberculous, suppurative, syphilitic meningitis (Onset often sudden, especially in the suppurative form, but also in tuberculous meningitis of nurslings. Monoplegia and hemiplegia often of a transitory nature. Hyperæsthesia, rigidity of the neck, recurring convulsions, fever, optic neuritis and paralysis of ocular muscles, vomiting, constipation, arrhythmic pulse).

B. (PREDOMINANTLY) CEREBRAL PROCESSES.

1. *Hydrocephalus* (Usually gradual onset, incomplete persistent paraplegia; often only symmetrical rigidity of the lower extremities, ataxia, signs of cerebral pressure, cranial deformity, atrophy of the ocular nerves).—Congenital or acquired; frequently preceded by (serous) meningitis; hereditary syphilis!
- [2. *Porencephaly, Microcephaly, Cerebral Hypertrophy* (Congenital conditions with psychic defects, divers manifestations of irritation).]
3. *Encephalitis* (Onset various; usually hemiplegia and monoplegia of the upper extremities, sometimes with cerebral disturbance, aphasia, and psychic defects; leading to characteristic positions of contracture; later on hemichorea and hemiathetosis; sensibility, sphincters intact).—After infections (diphtheria, influenza, measles, pneumonia, whooping-cough, pyæmia, syphilis); after trauma. Cerebral abscess (Usually pus fever, choked disk and other signs of cerebral pressure, but no bradycardia; often convulsions).—Traumatism, petrous bone affections, pyæmia. Acute policephalitis (Displaying the manifestations of an acute infectious disease, with convulsions, vomiting, fever; only from the first to the third year of age).
4. *Embolism [and Thrombosis] of a Cerebral Artery* [cerebral hæmorrhage], usually in the centrum ovale, in the capsule; less often meninges, cortex, pons, bulbus involved) (Sudden onset with alarming manifestations, but without fever, sometimes with convulsions. Usually hemiplegia with paralysis of the facial and hypoglossal. Paralysis at first usually flaccid, later on spastic, distinctly pronounced;

B. (PREDOMINANTLY) CEREBRAL PROCESSES.—Continued

- arm more affected than leg, hand more than arm;
often sensory and trophic disturbances; growth of
bones backward; frequently symptoms of irritation
(chorea and athetosis) later on in the paralyzed parts):
- (a) Cardiac affections, also simple dilatation (contracted kidneys!): Hypertrophy and valvular insufficiency.
 - (b) Traumatisms, also birth trauma: internal cephalæmatoma.
 - (c) Burns, intoxications.
 - (d) Hæmorrhagic diathesis; also leukæmia, sepsis, syphilis.
 - (e) Acute infectious diseases: Whooping-cough, (Cough attacks or toxic effect), diphtheria, pneumonia, typhoid, scarlet fever, measles.
 - (f) Other cerebrospinal affections, meningitis, sinus thrombosis.
5. *Cerebral Tumor* (Gradual beginning, spastic monoplegia and hemiplegia together with paralyses of cerebral nerves, the latter generally multiple*, different according to localization; no distinct atrophy; signs of cerebral pressure; vomiting, bradycardia, choked disk and later optic atrophy, headache).—Tuberculosis, syphilis, sarcoma, glioma, cysts, etc.
6. *Diffuse Cerebral Sclerosis* (Congenital or before the third year, certainly before the sixth; sometimes insidious, often sudden commencement with convulsions, which will continue; hemiplegia, paraplegia, tetraplegia. Condition after a time stationary, even somewhat susceptible to improvement; slight atrophy of soft parts and skeleton; usually mental, seldom sensitive disturbances. Sphincteric paralysis).—Hereditary and birth defects of various kinds. Hereditary syphilis.
7. *Multiple, Cerebrospinal Sclerosis* (Usually after the third year of life; onset gradual or sometimes by spurts; but always slowly progressive. Spastic paraplegia, at first intermittent; often more stiffness, ataxia, and awkwardness than paralysis. Tottering walk, scanning speech, intention tremor, nystagmus, mental functions long maintained).

[8. *Amaurotic Family Idiocy* in the final stage.]

C. SPINAL AFFECTIONS.

1. *Transverse Myelitis* (Commencement differs according to the character of the affection, in hæmorrhages sudden,

*Exception: Isolated ptosis in cerebral gumma.

C. SPINAL AFFECTIONS.—*Continued*

otherwise more gradual; paralysis usually symmetrical. Always sphincteric paralysis).—Focal affection of the spinal column; hæmorrhages, birth and other traumatism, postinfectious myelitis:

- (a) Superior cervical myelitis (Spastic paralysis of the upper and lower extremities, together with paralysis of neck and diaphragm and bulbar signs. Incontinence with overfilled bladder).
 - (b) Inferior cervical myelitis (Spastic paralysis of the upper extremities, together with flaccid paralysis of the lower. With oculopupillary signs and paralysis of the trunk muscles. Incontinence with overfilled bladder).
 - (c) Dorsal myelitis (Spastic paralysis of the lower extremities (upper free!), segmentary anæsthesia of the lower abdominal half; decubitus, difficult expiration; incontinence with overfilled bladder).
2. *Unilateral Paralysis of Brown-Sequard* (Unilateral paralysis with increased reflexes and loss of the sense of position; on the other side, anæsthesia for all qualities of susceptibility, with the exception of the feeling of position).—Tumors [injuries].
 3. *Spastic Spinal Paralysis* (Occurrence from eighth to fifteenth year; setting in slowly, progressive, ascending; spastic paraplegia, with characteristic position of the legs, crossing of thighs and stilted walk with body bent forward; more spasm than paralysis, the former increased by irritation; no atrophy, no sensory or psychic disturbances, no sphincter paralysis; healthy children affected; often occurring in families).—Usually syphilitic, sometimes after acute infectious diseases: diphtheria, influenza, polyarthritis, whooping-cough. Similar syndrome transitory in myelitis, multiple sclerosis.
- Little's disease (Picture similar to spastic spinal paralysis, but traceable to intra-uterine or birth injuries, therefore occurring rather early, in premature birth, asphyxia; with signs of a simultaneous cerebral affection; psychic defect, disturbances of ocular muscles, sometimes choreic and athetotic movements; becomes stationary).
4. *Amyotrophic Lateral Sclerosis* (Gradual onset (never before the tenth year) in the arms; later in the legs and then the bulb becomes involved: in the later stage very distinct atrophy, also perhaps RD., but always rigidity of

C. SPINAL AFFECTIONS—*Continued*

the muscles. No disturbances of sphincters, mind, nor sensibility).

D. HYSTERIA (Occurs almost never before the sixth year; often setting in suddenly, accompanied by contractures difficult to overcome, spastic hemiplegia, monoplegia, or paraplegia. Cerebral nerves mostly (facial and hypoglossal always) free; often rapid change of the signs, atrophy attacks only the soft parts).

166. CONDITIONS OF FLACCIDITY AND NON-USE OF EXTREMITIES, WITHOUT TRUE PARALYSIS

(flaccid pseudoparesis and pseudoparalysis)

A. WITH PRIMARY PAINFUL CHANGES OF THE BONES AND JOINTS (Electrical excitability normal, no degenerative muscular atrophy, no trophic or sphincteric disturbances; "pain paralysis").

1. Parrot's pseudoparesis (Occurring gradually in children between the first and third months of life; especially the humerus affected, very frequently also the radius and ulna, less often lower extremity; circular swelling at the distal end of the diaphysis, with crepitation and pain; at first only one bone is involved, later several; characteristic position of the upper extremity in humerus affection: arm hangs down relaxed, pronated; fingers move. Lower extremity in femoral affection: leg relaxed, slightly bent and rotated outward, toes move. Sometimes preceded by apparent trauma; sometimes other characteristic signs of syphilis).—Hereditary syphilis (syphilitic osteochondritis).
2. Rachitis (In children in the second half-year or second year, gradually developing weakness of the lower extremities with disinclination to use them; in the dorsal position complete mobility of the legs; drawn up as the child is lifted; epiphyseal enlargements may still be completely absent; affection never unilateral, usually quite symmetrical; usually, upper extremities also involved).
- [3. Osteomalacia, myxœdema, mongoloid.]
4. Infantile scurvy (Onset rather sudden in children from the second to the fifth half-year; violent pains on taking hold of the thickened lower femoral epiphysis, as well as of other osseous ends; fever; joints not involved; affection generally bilateral, but in different degrees and periodically intermitting. Skin normal or with traces of œdema. Upper extremity seldom affected and always in a less degree).

A. WITH PRIMARY PAINFUL CHANGES, etc.—*Continued*

5. Luxations and fractures (Sudden development or congenital presence; careful examination of the bones!).
6. Osteomyelitis (Almost exclusively older children; high fever, pain, swelling, radiogram!).
7. Osteosarcoma (Generally thigh, radiogram!).
8. Rheumatic polyarthritis (Seldom in little children; *see Articular Effusion*, No. 203).
9. Gonorrhœal polyarthritis. Periarticular phlegmon.

B. IN PRIMARY AFFECTIONS OF THE MUSCLES.

1. *Myopathic Forms of Progressive Muscular Atrophy* (Rare in first childhood, generally occurring toward puberty; setting in gradually, very slowly progressive weakness of the muscles; symmetrical; deep reflexes retained in the beginning, disappearing later; no fibrillary twitchings; excitability of nerve and muscle diminished, but no RD., no pronounced sensitive disturbance; occurs in families):

(a) Pseudohypertrophic (Duchenne) type. (Commences in lower extremities; awkwardness and rapid fatigue in walking, balancing of the trunk; trunk, arm, shoulder later involved; "climbing up one's self," pelvic stoop; pes equinus with contracture, lordosis; volume of the affected muscles often normal or materially increased).

(b) Scapulohumeral (Erb) type (Commences in arm, shoulder, seldom back, leg; later lumbar muscles especially involved, quadriceps, peroneus; sternomastoid, deltoid muscle, distal muscles of the upper extremities free; lordosis, dawdling walk, muscle volume generally not increased).

(c) Facioscapulohumeral (Landouzy-Déjerine) type (Commences in the face, later sometimes the shoulder and pelvic girdles and extremities; peculiarly dull, rigid facial expression, mask-face; inability to laugh, whistle, to close the eyes; muscle volume diminished).

2. *Muscular Traumatisms*.C. IN NEUROSPINAL AFFECTIONS. (Transition to genuine paralysis, *see* No. 164).

1. *Neurospinal Forms of Progressive Muscular Atrophy* (Insidious commencement; progressive weakness of certain muscle groups with much atrophy; symmetrical; electrical excitability always diminished, and usually RD.; almost always occurs in families):

G. IN NEUROSPINAL AFFECTIONS, etc.—Continued

- (a) In early infancy, Hoffman-Werdnig type (Beginning in first year of life: pelvis, loins, and thighs (glutæus, triceps); inability to keep erect; walking is forgotten or never acquired at all; nucha, neck, arm later affected; centrifugal progression to hand and foot; frequently fibrillary twitchings; lordosis).
- (b) Charcot-Marie type (Beginning generally in feet and legs, after the fourth year; paralytic pes valgus; dragging walk; later, upper extremities: claw hand; centripetally progressing, but trunk, shoulder, face free; seldom fibrillary twitchings and sensory disturbances).
- (c) Déjerine-Sottas type (Beginning usually after the fifth year in the lower extremities (perone, anterior tibial, extensors); pes equinus, foot strongly arched, often fibrillary twitchings; ataxia, lancinating pains: pseudotabes; Brach-Romberg's sign. Myosis and nystagmus; reflex immobility of the pupil. Kyphoscoliosis).

2. *Nerve Traumatisms*, as in paralysis brachii dolorosa (see *Pareses*).

[3. Friedreich's hereditary ataxia.]

D. FUNCTIONAL NEUROSES AND CONDITIONS OF A DOUBTFUL NATURE.

- [1. Amaurotic family idiocy (Laxity of the entire musculature, increasing steadily until death in the second or third year; visual disturbances and typical ophthalmoscopic finding; family occurrence among Jews).]
- 2. Also other forms of idiocy (mongoloid, etc.).
- [3. Congenital myotonia, Oppenheim (Observed in the first 2 years of life; laxity of the leg muscles, absence of the patellar reflex; diminished electrical excitability; unre-sisting joints; spontaneous tendency to cure; cause?).]
- 4. Hysteria (Abasia, astasia, often with full ability to move in the dorsal position).
- 5. Chorea minor mollis (Before or with the typical chorea Sydenham).

167. DIMINUTION AND LOSS OF CUTANEOUS SENSIBILITY

A. UNIVERSAL.—Idiocy (early symptom!); agony, coma, especially after cerebral affections.

B. LOCALIZED.

- 1. Affections of peripheral nerves (Very seldom partial paralysis of sensation; generally diminution or absence of the

B. LOCALIZED.—Continued

deep and superficial reflexes; localization corresponds to the area of extension of the affected nerve; usually no trophic disturbances): Neuritis (lead!), neuralgia. Traumatic paralyses of plexus and nerves.

2. Spinal affections (Frequently partial paralysis of sensation, often increase of the reflexes, and trophic disturbances): Leptomeningitis and spinal pachymeningitis, hyperæmia and spinal hæmorrhage. Myelitis (especially in Pott's disease), acute anterior poliomyelitis I, tumors of the spinal cord, spina bifida. Multiple sclerosis. Infantile tabes. [Syringomyelia.]
3. Cerebral affections (Sometimes partial paralysis of sensation, frequently hemianæsthesia): Tumors and hæmorrhages (especially optic thalamus, centrum ovale, and parietal cortex).
4. Functional neuroses (Frequently partial paralysis of sensation and hemianæsthesia): Hysteria, traumatic neurosis, epilepsy (in and after the paroxysm), [tetany].

**168. TENDERNESS, HYPERÆSTHESIA IN VARIOUS AREAS
OF THE BODY**

1. *General (or very Extensive) Cutaneous Hyperæsthesia:*
 - (a) Spinal or cerebrospinal meningitis (especially epidemic and suppurative meningitis; less often tuberculous meningitis), and hæmorrhages of the spinal cord.
 - (b) Certain acute infectious diseases, especially influenza, typhoid, and miliary tuberculosis.
 - (c) Hysteria.
2. *Hyperæsthesia of Nerve Trunks.*—Neuralgia, neuritis; psychogenic affections.
3. *Hyperæsthesia of the Spinal Cord:*
 - (a) Nearly all conditions that lead to rigidity of neck and opisthotonos, *see No. 136*, especially spondylitis and myelitis; spinal tumors; also hysteria, exophthalmic goitre and chorea.
 - (b) Rheumatism of the vertebral articulations and dorsal muscles.
 - (c) Neuralgia of the spinal nerves.
4. *Sensory Hyperæsthesia* (Photophobia, odorophobia, dread of noise):
 - (a) Meningitis, especially cerebrospinal meningitis.
 - (b) Conjunctivitis before infectious diseases.
 - (c) Migraine; psychogenic conditions.
 - (d) Amaurotic family idiocy.

169. HEADACHE *

Apparent in the youngest babies: pounding the head, pulling the hair, wrinkling the eyebrows.

Very frequently accompanied by vomiting!

A. ACCIDENTAL.

1. *Affections of the Cerebrospinal System*, especially with cerebral pressure (Often of great intensity, not corresponding to temperature curve, augmented by sensory irritations; together with other cerebrospinal signs):
 - (a) All disturbances of circulation in the brain: Anæmia, active and passive hyperæmia. Heat stroke, sinus thrombosis, sudden cardiac weakness. Violent coughing attacks, concussion.
 - (b) Meningitis.
 - (c) Cerebral tumor (Partly sharply localized, narrowly confined, sometimes paroxysmal, not susceptible to medication; susceptibility to cranial percussion, progressive.)
 - (d) (Polio-) encephalitis and poliomyelitis.
 - (e) Cerebral abscess (often localized, increased through straining, coughing!).
 - (f) Syphilitic sclerosis (pains at night, often hemicranial), tabes dorsalis.
 - (g) Embolism and cerebral hæmorrhages.
2. *Functional Neuroses* (here partly accompanying the attacks: Chorea, tetany, epileptic aura).
3. *Acute Infectious Diseases* (nearly all those with sudden onset; here often prodromal): Typhoid, influenza, malaria, diphtheria, pneumonia; also non-specific angina. Acute poliomyelitis and poliencephalitis.
4. *Acute Intoxications*:
 - (a) Ectogenous: Very many poisons (digitalis, ergot, quinine, opium, carbonic oxide, alcohol, etc.), tainted articles of food, indigestion (here headache along with gastritis, etc., icterus, vomiting!).
 - (b) Endogenous: Uræmia in acute incipient nephritis, also perhaps in intermittent albuminuria.

B. HABITUAL (Without organic changes of the cerebrospinal system; changes of the circulation, disturbances of nutrition, vasomotor affections, endogenous chronic toxicoses?).

1. True migraine (Often unilateral, but also frontal, symmetrical; paroxysmal with vomiting, somnolence, pallor,

*Aside from that in affections of the cranial bone (syphilis), and of the soft cranial walls (abscess), in nasal and frontal sinus, catarrh, nasal polypus, otitis.

B. HABITUAL, etc.—Continued

spastic yawning, general sensory hyperæsthesia, especially photophobia; attacks are always separated by a few free days, few attacks lasting 24 hours; often periodical).

2. Conditions similar to migraine (Vague, remittent pains without nausea, vomiting, and photophobia, with sleeplessness and pavor nocturnus; there are no entirely free intervals and no long and regular pauses; usually every day; therapeutically influenced by absence from school and change of manner of living).—Constitutional factors: "Neuro-arthritis," neurasthenia, hysteria; homologous or heterologous nervous hereditary taint, degeneration; anæmia, chlorosis. Exciting factors: Astigmatism, chronic nasal affections, adenoid vegetations; chronic vulvovaginitis, orthostatic albuminuria, dyspepsia, intestino-atic constipation, worms, goitre, mental overwork.

G. FORMS INTERMEDIATE BETWEEN THE GROUPS A AND B.

1. *Neuralgia in the Area of the Head* (Often unilateral, occurring periodically; intensity slighter than in adults; typical pressure points; sensory, motor, and vasomotor concomitant manifestations; paræsthesia, rigidity, herpes, urticaria):
 - (a) Ophthalmic neuralgia; nerve V, branch 1 (Pressure point at supraorbital foramen; hyperæmia of the lids, and lachrymal secretion).
 - (b) Supramaxillary neuralgia; nerve V, branch 2 (Pressure point at infraorbital foramen; "toothache").
 - [(c) Inframaxillary neuralgia; nerve V, branch 3 (Pressure point at mental foramen; coated tongue, salivation).]
 - (d) Occipital neuralgia; cervical nerve II, posterior branch (Pressure point at great occipital nerve; rigidity of neck).
2. *Mechanical* (trauma), *Infectious* (influenza, typhoid, malaria, syphilis), *Toxic* (metals), and *Rheumatic Injury* of the Nerves; same factors as in neuritis; especially in neuropathic, arthritic, anæmic, and hysterical individuals. Also in nasal foreign bodies and coryza; sinus thrombosis (sinus cavernosus).
3. *Progressive Paralysis* (initial symptom), *Friedreich's Ataxia*.
4. *Diabetes Mellitus*.
5. *Hysteria* ("clavus").

170. VERTIGO

The same conditions that lead to headache; of the cerebral affections, especially those of the cerebellum; of the sensory organs, especially those of the inner ear.

171. NEURALGIA-LIKE PAINS

Mostly spontaneous.

A. IN THE THORACIC REGION.

1. Affections of the thoracic viscera: Tracheitis, pulmonary tuberculosis, apex phthisis, miliary tuberculosis, pleuritic exudate (Often only pain on pressure, as for instance in percussion), pleuritic adhesions at the apex (Especially in tuberculosis), incipient croupous pneumonia (Usually unilateral), cardiac affections, especially pericarditis (With oppression, radiating toward the back and into the shoulder), swelling of the lymphatic glands, especially of a tuberculous nature, also in Pfeiffer's glandular fever (In the region of the sternum). Oesophageal affections, especially stricture; traumatic and rheumatic affections of the diaphragm (Localized in the form of a girdle). [Aortitis.]
2. Affections of the osseous thoracic wall: Osteomyelitis, periostitis of the sternum; caries, fracture of the ribs. Various anæmias (sternal pains).
3. Rheumatism of the pectoral muscles (Pains in the muscle itself, greatly increased on moving trunk and arm, often originating suddenly, sciatica).
4. Intercostal neuralgia (Pressure points in the anterior median line, by the side of the spinal column, in the axillary region, frequently along with herpes zoster).—Especially at puberty, after dyspepsia; see also under *Headache*, No. 169, C.
5. Hepatic affections (*e.g.*, abscess) (Radiating toward the shoulder).

B. IN THE ABDOMINAL REGION.

1. Affections of the abdominal viscera (*see Abdominal Pains*, No. 55), frequently radiating toward the abdominal wall—for instance, in appendicitis, into the region of the first dorsal nerve.
2. Pains of the abdominal muscles in violent cough, straining.
3. Lumbo-abdominal neuralgia in Pott's disease, etc.

C. IN THE LUMBAR REGION.

1. Onset of acute infectious diseases (variola, typhoid, influenza).

C. IN THE LUMBAR REGION.—*Continued*

2. Spinal affections, especially spinal meningitis (cerebrospinal meningitis and tuberculosis with spinal localization).—Transverse myelitis, spina bifida (occulta), Pott's disease, hæmorrhages, tumors (Girdle pain, radiating toward the extremities).
3. Renal affections, especially nephritis, pyelitis, hæmorrhagic kidney infarction, nephrolithiasis (Violent paroxysmal cutting pains from the renal region toward the abdominal walls, toward the bladder, the rectum, and the genitals; paroxysms often occasioned through traumatisms and concussions).
4. Coccygodynia.—Hæmorrhoidal pains. Insufficiency of cardiac valves.

D. IN THE EXTREMITIES (especially the lower).

- [1. Ischias (Pressure points in the incisura ischiadica, at the lower border of the glutæus and in the popliteal space; muscular spasms of the calf, and paresis).—Neuralgia and neuritis of the ischial nerve.]
2. Achillodynia, metatarsodynia.
3. Affections of the spinal cord: Tabes with paralysis, Friedreich's ataxia; progressive spinal muscular atrophy, Pott's disease, myelitis, tumor, etc.
4. Radiating pains in abdominal affections, such as appendicitis, etc.
5. Thrombosis of the femoral veins.
6. Incipient tuberculous coxitis and other articular and osseous affections.

172. STRABISMUS, SQUINTING

(faulty position of the whole eye—strabismus in a broad sense—strabismus sensu lat.)

1. *Paralytic Squinting* (The deviation is shown only in movements—or positions—in the tract of the paralyzed muscle, and grows with the excursion: squint angle inconstant; secondary squint angle larger than the primary; false projection, pathological position of the head, vertigo, etc.).—Occurrence, *see Tables belonging to Nos. 172 to 176.*

2. *Spastic Squint*:

- (a) Clonic = nystagmus.—Congenital and early acquired weak-sightedness; leucoma after blennorrhœa, retinitis, etc.; otherwise, for occurrence, *see Tables Nos. 172 to 176.*
- (b) Tonic (Behavior similar to 1, above), but intermittent and usually part manifestation of general tonic convulsions).

3. *Concomitant, True Squint; Strabismus* (Deflection of the squinting eye is in all movements alike: squint angle constant; secondary

squint angle equal to the primary one. Diminution of the visual acuity; no false projection).—For occurrence and importance, see ophthalmological text books.

4. *Mechanical Squint*.—Orbital affections: depression of the orbital roof in chronic hydrocephalus. (Direction of look in both eyes habitually downward to the extreme.)

NOTE.—In the first few weeks of life healthy children show now and then incoördinate movements of the eyeballs: association of the binocular visual act not yet fixed.

PARALYTIC SQUINT

A. NERVE AFFECTED.

Eye-ball deflected.	Head (often) held by way of compensation toward the	Movement of eye-ball limited.	Accompanying signs.	Nerve affected.
Inward paralytic convergent strabismus.	Affected side.	Outward.		Abducens.
Outward (and a little inferiorly), paralytic divergent strabismus.	Healthy side (and a little upward).	Inward, upward, and decidedly downward.	Paralytic exophthalmus, mydriasis, reflex immovability of pupil, paralysis of accommodation.	Oculomotorius (total paralysis).
Inward and upward, paralytic convergent strabismus.	Affected side and downward.	Outward and downward.		Trochlearis.

B. PRESUMABLE PLACE OF ORIGIN OF THE PARALYSIS AND ITS MOST FREQUENT CAUSES.

Character of the paralysis and the accompanying circumstances.	Place of origin.	Most frequent causes.
(a) Paralysis with signs of orbital affection; usually only paresis; combined paralysis.	Orbit.	Trauma, osseous affection, tumor, cold.
(b) No orbital signs; frequently paralysis isolated or combined; rarely partial paralysis; disturbance only in a few muscles supplied by the oculomotor nerve; progressive involvement of other cerebral nerves (including the optic).	Base of brain.	Various forms of meningitis; syphilitic meningitis, fracture of base of skull, rachitis, basilar tumors.
(c) No orbital signs; unilateral partial paralysis (of the oculomotor nerve), sometimes combined with alternating hemiplegia (and isolated paralysis of the facial nerve).	Crus cerebri and neighborhood.	Cerebral tuberculosis, tumor (glioma); syphilis, and the causes mentioned under (a).
(d) No orbital signs; bilateral (or unilateral) partial paralysis (of the oculomotor nerve); combined with hemiplegia of the same side or paralysis of the hypoglossus and trigeminus. (a) Internal ophthalmoplegia. Mydriasis, paralysis of accommodation. (b) External ophthalmoplegia; combined paralysis of the external oculomotor muscles.	Nuclear region.	Acute processes: Hæmorrhages from milary aneurisms after endarteritis in intoxications, infection (syphilis, influenza, diphtheria), trauma. Embolic softening foci; acute superior poli-encephalitis. Chronic processes: Progressive paralysis of the ocular muscles, sclerosis, tumors, myelitis, tabes, chronic bulbar paralysis, Little's disease.
(e) Associated motor insufficiency, disturbance of simultaneous function of both eyes, conjugate deviation, reflexes normal.	Cortex and sub-cortical medullary layer.	Apoplexy, embolism, with encephalomalacia (syphilis, renal and cardiac affections, intoxications), tumors.

CHANGES IN THE SIZE OF THE PUPILS

173. MYDRIASIS (Pupils wider). (Toxic effect of carbon dioxide, of atropine, strychnine, cocaine, and kindred poisons).	174. MIOSIS (Pupils narrower). (Toxic effect of pilocarpine, eserine, morphine, muscarine, and kindred poisons).
A. SPASTIC ¹ : Spasm of the musculus dilatator pupillæ (iris) (sympathetic nerve). B. PARALYTIC : Paralysis of the musculus sphincter pupillæ (oculomotor nerve). For special occurrence, see <i>Tables belonging to Nos. 172 to 176</i> .	A. SPASTIC : Spasm of the musculus sphincter pupillæ (oculomotor nerve). B. PARALYTIC : Paralysis of the musculus dilatator pupillæ (iris) (sympathetic nerve). For special occurrence, see <i>Tables belonging to Nos. 172 to 176</i> .
C. FROM ORBITAL CAUSES : See special works.	C. FROM ORBITAL CAUSES : See special works.
D. REFLEX INFLUENCE : 1. Certain peripheral irritations. ² 2. Intestinal parasites. 3. Psychical irritations. 4. Pain, terror, sexual excitement.	D. REFLEX INFLUENCE : 1. Angle of incidence. 2. Convergence of the visual axes.
E. ABOLISHING OF THE PHYSIOLOGICAL REFLEX TONUS in interrupted conduction of centripetal part of optic-pupillary reflex tract. (Both pupils uniformly dilated, reflex immovability of pupils. Reaction of convergence maintained).	

¹Usually transitory; also in irritation of the sympathetic nerve in affections of the bronchial glands and by exudative pleuritis.

²Absence of this reflex is said to be an early symptom of tuberculous meningitis, as is mydriasis with backward bending of the head.

CHANGES OF THE LID FISSURE

175. LAGOPHTHALMUS (Abnormally wide). (Inability to (completely) close the lid fissure).	176. PTOSIS (Abnormally narrow). (Inability to (completely) open the lid fissure. Simulated by certain affections of the lid).
A. SPASTIC : Spasm of the musculus levator palpebræ superioris or musculus Müllerii (oculomotor nerve, sympathetic nerve). B. PARALYTIC : Paralysis of the musculus orbicularis orbitæ (facial nerve). For occurrence, see <i>Tables to Nos. 172 to 176</i> .	A. SPASTIC : Spasm or hypertonicity of the musculus orbicularis orbitæ (facial nerve). B. PARALYTIC : Paralysis of the musculus levator palpebræ superioris or musculus Müllerii (oculomotor nerve, sympathetic nerve). For occurrence, see <i>Tables to Nos. 172 to 176</i> .
[C. MECHANICAL, in exophthalmus, tumors, etc.]	[C. HABITUAL, in congenital dysplasia of the musculus levator palpebræ.]

177. SEE TABLE ON FOLLOWING PAGE

178. EARACHE

Acute external otitis and otitis media in infectious diseases (measles, scarlet fever, diphtheria, influenza, mumps, whooping-cough, typhoid, erysipelas, lobar pneumonia, cerebrospinal meningitis, varicella, hereditary syphilis, infectious intestinal conditions, diverse forms of acute rhinitis and angina), in foreign bodies in the exterior auditory canal.

Chronic otitis after acute affections; also in tonsillar hypertrophy, adenoid vegetations, scrofulosis, tuberculosis.

177. SURVEY OF A FEW EXTRA-ORBITAL AFFECTIONS WITH OCULAR SYMPTOMS OF IMPORTANCE FOR DIAGNOSTIC PURPOSES

Blepharitis.....	Lymphatism, scrofulosis.	Exophthalmus— <i>Cont.</i>	Mumps.
Fissures at the angles of the eye.	Hereditary syphilis.		Chronic arthritis.
Œdema of the lids....	Nephritis, cardiac affections, sinus thrombosis, affections of the eye-ball and conjunctiva.		Stenosis of the upper air tracts (nose, pharynx, larynx, trachea).
Blepharospasm.....	Hysteria, spasms nutans, local ocular affections.	Atrophy of the visual nerves.	Irritation of sympathicus.
Long cilia.....	Habitus tuberculosus.		Diffuse and multiple sclerosis, tabes and paralysis, hydrocephalus and microcephalus (all on syphilitic basis). Friedreich's ataxia neuroglioma.
Lid fissure rising outward, markedly arched eyebrows.	Mongoloid idiocy, degeneration.	Optical neuritis and papillitis.	Chronic hydrocephalus, encephalitis, cerebral abscess, acute meningitis, polyneuritis in acute infections running a grave course, intoxications (lead); cerebral syphilis.
Epicanthus, with thickened lids and narrow lid fissure.	Mongoloid and myxœdematous idiocy, degeneration.	Choked disk.....	Hydrocephalus, chronic meningitis, cerebral tumors and abscesses, sinus thrombosis, cerebral syphilis.
Simple conjunctivitis..	Nasal affections, neuralgia; acute infections: measles, whooping-cough, German measles, influenza, cerebrospinal meningitis, [scarlet fever].	Albuminuric retinitis.	Chronic parenchymatous and interstitial nephritis.
Blennorrhœic conjunctivitis.	Gonorrhœa.	Œdema of the retina.	Thrombosis sinus cavernosus.
Pseudomembranous conjunctivitis.	Diphtheria and non-specific chronic affections.	Retinal hæmorrhages.	Sepsis neonatorum (with embolisms), typhoid, whooping-cough, leucæmia, pernicious anæmia.
Eczematous conjunctivitis.	Scrofulosis.	Typical retinal changes.	Leucæmia, amaurotic idiocy.
Vesicular conjunctivitis.	Varicella. Influenza.	Hemianopsia.....	Infantile cerebral paralysis, cerebral tumors, etc.
Conjunctival papule.	Syphilis, hæmorrhagic diathesis, whooping-cough, trauma, tuberculosis, scrofulosis.	Inability of fixation.	Early symptom of idiocy.
Conjunctival hæmorrhages.		Amblyopia, amaurosis without objective ocular changes.	1. Toxic: ectogenous (extra-utero filicis maris, antonin, lead); endogenous (uræmia, diphtheria, and typhoid).
Phlyctenular keratitis.			2. After severe hæmorrhages and anæmic affections.
Parenchymatous keratitis.	Syphilis, late hereditary.	Photophobia.....	3. Hysteria.
Iridocyclitis.....	Cerebrospinal meningitis, typhoid, scarlet fever, syphilis.		Conjunctivitis, as in measles, migraine, quinine and other intoxications.
Panophthalmitis.....	Gonorrhœa, syphilis, injuries, etc.	Xanthopsia.....	Santonin poisoning, icterus.
Chorioidal tubercle...	Miliary tuberculosis and tuberculous meningitis.		
Exophthalmus.....	Complete oculomotor paralysis, exophthalmic goitre, engorgement of blood or lymph (and hæmorrhage) behind the eye-ball and in the cervical region (sinus thrombosis, fracture of base, whooping-cough, infantile scurvy, asphyxia, adenoid vegetations), myxœdema.		

179. TINNITUS AURIUM

1. Otitis.
2. Disturbance of cerebral circulation.
3. General anæmic conditions, hypertrophy of the tonsils, intoxications (salicylic acid, antipyrin, quinine), toxicoses (uræmia).
4. Intestinal parasites, masturbation, goitre.

180. SYNDROME OF CEREBROSPINAL IRRITATIONS AND PRESSURE

Most important signs (singly or combined, generally occurring after one another):

	Psychical.	Motor Sphere.	Sensory Sphere.	Vasomotor Sphere.
Irritation.	Unrest, excitation, delirium, carphology, sighing.	Twitching, trembling, rigidity of muscles, spasms of single groups of muscles: rigidity of neck, opisthotonos, gnashing of teeth, scaphoid belly, general convulsions, Kernig's phenomenon, arrhythmic pulse, bradycardia, cerebral vomiting, enterospastic constipation. (Irritation of the splanchnic area.)	Cephalalgia, general hyperæsthesia.	Increased flow to the skin, precipitate change of color, Trousseau's spots.
Pressure.	Sleeplessness, stupor.		Tinnitus aurium, vertigo.	

Frequently subsequent or concurrent signs :

	Psychical.	Motor Sphere.	Sensory Sphere.	
Paralysis.	Coma, stupor.	Paralysis of single nerves and muscles, (especially of the eye); general central paralysis, tachycardia, Cheyne-Stokes respiration.	Anæsthesia.	

NOTE.—In distinguishing the various causative conditions which are now being enumerated, the result of lumbar puncture plays an important rôle. (See No. 181.)

A. ORGANIC CHANGES IN THE CEREBROSPINAL AREA.

1. *Meningitis* (Usually manifold symptoms, marked constipation, scaphoid belly, sighing, crying out, gnashing of teeth, pulse anomalies, change in complexion, bulging and tension of fontanelle in (younger) children. Optic neuritis. Usually fever and established course, differing according to nature of affection):

- (a) *Tuberculous leptomeningitis* (By far the most frequent form! Almost always gradual commencement, long continued, uncertain prodromes, headache moderate; apathy gradually increased to coma; beyond the first year of life abdomen sinks in in spite of constipation; arrhythmic pulse, bradycardia very pronounced, distinct basal—usually few spinal—manifestations; subfebrile or even afebrile course [chorioid-tubercle]; other specific pathological foci! Diagnosis of tuberculosis ex juvantibus!).—

A. ORGANIC CHANGES IN THE CEREBROSPINAL AREA.—*Continued*

Often after measles, whooping-cough, and operative interference in tuberculous foci.

- (b) Simple serous leptomeningitis (Almost exclusively in the first and second years of life, onset rather rapid; irregular fever curve or sometimes absent; pressure symptoms at first prominent; tension of fontanelle, enlargement of cranium, choked disk; opisthotonos, laryngospasm, frequently recurring convulsions; prolonged course with remissions).—In the course of gastro-intestinal and bronchopneumonic affections. In aural affections; as angioneurosis (?), in insolation, concussion, poisoning, certain anæmic conditions, etc. Perhaps also in infectious diseases; measles, pertussis, influenza, syphilis.
- (c) Cerebrospinal leptomeningitis (occurring sporadically and epidemically) (Usually sudden onset with chills and convulsions; violent rigidity of the neck and opisthotonos, Kernig's sign, vomiting, pain on pressure of the spinal column, general cutaneous and sensory hyperæsthesia; seldom arrhythmic pulse and bradycardia; fever, manifestations of paralysis, and loss of consciousness not prominent. Accompanying frequently: herpes, roseola, articular affections. Usually marked remissions, frequent intermissions, course prolonged, but relatively frequently favorable).
- (d) Suppurative leptomeningitis (Usually sudden onset, with alarming manifestations; high fever, tempestuous course; delirium and convulsions prominent, hyperæsthesia of the skin; Kernig's sign; basal manifestations less distinct, especially less disturbance of ocular muscles, no optic neuritis, usually no bradycardia nor irregular pulse. Considerable leucocytosis. In nurslings, often neither grave general nor distinct local symptoms).—Trauma (*e.g.*, ventricular puncture) and heat stroke. Extension of the inflammatory process to adjacent tissues. Otitis, mastoiditis, ozæna, erysipelas, phlegmon, retropharyngeal abscess, thrombophlebitis. Eroded meningocele. General septicæmia in pneumonia, typhoid, influenza, polyarthritis, and other infections of unknown origin.
- (e) Syphilitic leptomeningitis (chronic basilar) (headache, vomiting, vertigo, convulsions and delirium,

A. ORGANIC CHANGES IN THE CEREBROSPINAL AREA.—Continued

paralysis of cerebral nerves; usually together with hemiplegia, hemianæsthesia, aphasia as signs of other syphilitic cerebral affection, generally of an encephalitic nature; chronic course spontaneously remittent; effect of specific treatment!).

(f) *Hæmorrhagic pachymeningitis* (Onset usually sudden, with eclampsia, of which spasms and contractures remain behind; pulse tense, frequent, regular; no constipation, always hæmorrhages at the fundus).—Syphilis, trauma, hæmorrhagic diathesis and acute infectious diseases, chronic disorders of nutrition.

2. *Cerebral Tumor* (Status tumorosus, stupor, fixed look, often Jacksonian epilepsy, pulse changes, localized headache, distinct choked disk; usually no fever, condition remaining long unchanged; occurs chiefly in older children).

3. *Encephalitis and Cerebral Abscess* (After a usually prolonged latency symptoms of irritation and pressure, especially pulse changes; vomiting and choked disk generally less pronounced, may remit and periodically be totally absent; headache varying; paralysis occurs comparatively early, not of a basal nature generally, but pointing to a circumscribed cerebral affection; monoparesis, aphasia, central paralysis of facial nerve, conjugate deviation). Trauma, infection, metastasis, extension of aural and nasal affections.

4. *Sinus Thrombosis* (Often sudden development, partly without fever; frequently characteristic manifestations of the cranial and cervical veins, facial œdema).—Sepsis, marasmus after diarrhœa, affections of the petrosal bone.

[5. *Acute Bulbar Paralysis*.]

B. DISTURBANCE OF CIRCULATION IN THE CEREBRAL AREA (Often normal or subnormal temperature, no focal manifestations, no paralysis, no optic neuritis nor choked disk).

1. *Cerebral anæmia* (Along with headache, tinnitus aurium, vertigo, vomiting, muscular twitchings, delirium and convulsions, involuntary defecation; also, fainting, somnolence, changes of the pupil, amaurosis, small, frequent, intermittent pulse, irregular respiration).

Hydrocephaloid (Only in nurslings after exhausting diseases; beside the above signs, characteristic position of arms and legs, fontanelle depressed, cranial bones displaced above one another, subnormal temperature).

B. DISTURBANCE OF CIRCULATION, etc.—Continued

2. Active and passive hyperæmia.—Trauma, insolation, whooping-cough, mental overexertion.

3. Concussion of the brain (Loss of consciousness, vomiting, bradycardia, ischuria, less frequently paralysis; history!).

C. "MENINGISMUS" (Usually only few signs present; these transitory; no pronounced local symptoms, fontanelle tense only in a spasm; seldom trismus and certain other localized spasms; seldom distinct pulse

FIG. 5.



First step in technic of lumbar puncture. Line drawn over the point of puncture from crests of the ilium. Palpation of the spinal process.

anomalies; never retinitis, papillitis—except albuminuric—no choked disk. Usually, the manifestations of the primary affection soon show themselves).

1. *Acute Infectious Affections*, especially prodromal in the "cerebral forms" of acute infections (Nearly all single signs occur, the total picture, however, is less complete. Rare or less pronounced are the pulse changes and the rigidity of the neck. Convulsions occur almost exclusively in the first years of life; paralysis, such as follows in meningitis, is absent; no optic neuritis; convulsions and

C. "MENINGISMUS," etc.—*Continued*

vomiting cease as the specific signs of the affection become evident; beside these there are high temperature and a typical temperature curve. Patellar reflex often diminished or absent): Croupous pneumonia, sepsis, typhoid,

FIG. 7.



Third step in technic of lumbar puncture. Removal of the cerebrospinal fluid.

FIG. 6.



Second step in technic of lumbar puncture. Attachment of manometer after successful puncture and removal of the mandrin.

scarlatina, influenza, erysipelas, osteomyelitis, whooping-cough, miliary tuberculosis, poli-encephalitis and myelitis, polyarthrititis (especially when the vertebral articulations are involved); also acute bronchopneumonia and gastro-enteritis of nurslings.

NOTE.—The possibility of complicating inflammation of the meninges is to be considered!

C. "MENINGISMUS," etc.—*Continued*2. *Acute Toxic Conditions* (Generally as in 1, but often afebrile).

- (a) Of ectogenous origin: Santonin, atropine, iodoform, alcohol, narcotics.
- (b) Of endogenous origin: Uræmia (May produce nearly all of the signs of meningitis except paralysis of the cerebral nerves with rigidity of the neck, scaphoid belly and constipation; as positive signs, albuminuric retinitis and grave nephritic changes of the urine). Intestinal autointoxication, cyclic vomiting (Sudden onset, with intestinal manifestations, acetonuria with frequent recurrence of the condition). Here belong also many cases of "febris ephemera, febricula." [Cholæmia, helminthiasis gravis.]

3. *Reflex Irritations* (Almost exclusively in the youngest children, unless there is a pronounced tendency to spasms on the basis of neuropathic constitution or hereditary taint; the original affection can always be established; aside from the recurring convulsions, the picture is for a long time pronouncedly pseudomeningitic; after removal of the cause all manifestations rapidly disappear).—Difficult dentition, gastric dyspepsia (faulty diet, coated tongue, meteorism, icterus, effect of therapy!), coprostasis, helminthiasis, otitis media and interna (here probably also the local affection spreads to the meninges or the circulatory disturbances in the same) (tenderness on pressure upon the tragus and the mastoid process, —often merely restlessness and rigidity of neck; sensorium free).

4. *Hysteria* (Patient generally over 10 years old; insufficient coördination of the existing morbid symptoms; exaggeration of some, such as headache, vomiting, strabismus, gnashing of the teeth; absence of others, such as fever, changes of the facial expression and general condition, paralysis of ocular muscle, paralysis of facial nerve, and optic neuritis. The convulsive signs are more distinct than the depressive; hyperæsthesia is confined to certain portions of the skin. Course irregular with remissions and intermissions).

NOTE.—Vomiting and headache alone (along with pulse changes) are also found in other conditions (see No. 169).

181. SEE TABLE FOLLOWING

181. SYMPTOMATOLOGY OF FLUID

Technic of Lumbar Puncture: The child sits at the edge of the table, where it is held in a bent-forward position, marks the upper limit of the puncture; namely, the horizontal line joining the iliac crests. The field of operation having lumbar spines and close below the finger-nail inserts a sterile needle about 1 mm. wide and fitted with a stylet, horizontally is suddenly diminished. The stylet having been withdrawn, the headpiece of the manometer tube is attached to the upper level of the mercury after this has ceased to rise.¹ Then the fluid is slowly drawn off into sterile glasses. The pressure

	Normal finding.		Tuberculous meningitis.		Cerebrospinal meningitis.		Suppurative meningitis.
	Infants.	Older children.	State of irritation and pressure.	State of paralysis.	Acute process.	Intermission.	
Pressure in mm. Hg. measured before drawing fluid; medium phase of respiration; patient sitting.	About 10-20.	About 15-25 [to 35].	Always considerably increased, often 50 mm. and beyond, sometimes excessive (100 mm. and more).	Not increased.	Usually increased, but not excessively (up to 50 mm.).	Normal.	In the beginning and at the climax usually increased up to about 40 mm.; never excessive.
Microscopic appearance of the fluid.	Clear as water; does not contain the finest "sun specks"; colorless; not even a yellowish tint in a thick layer.	Often clear (of the cases); otherwise, permeated by finest specks, glittering in the sunlight.	Slightly dense grayish white turbidity; very rarely purulent (mixed infection).	Always turbid; sometimes tinged with blood, seldom purulent.	Almost (or quite) clear.	Usually considerable grayish white or grayish yellow turbidity in yellow-tinted fluid (only quite exceptionally clear).	
Change of fluid on standing.	No change.	Very often formation of a cobweb-like coagulum; seldom formation of cloudy flakes.	Coagulation.	Sedimentation of pus, frequently formation of fibrin net.	No change.	Sedimentation of creamy pus and net formation.	
Character of cellular constituents, obtained by sedimentation. ²	Exceedingly few lymphocytes, erythrocytes and endothelia; no more than about 5 cells per c.mm.	Abundant lymphocytes and polynuclear leucocytes; the former often vastly preponderating, the latter increased in mixed infection.	Polynuclear cells predominating, lymphocytes in the stage of advanced healing, possibly erythrocytes.		Polynuclear cells predominating.		
Albuminous contents of the fluid "A," determined after Brandberg.	0.02% ≤ A < 0.05% (Globulin).	Always increased: 0.1 - 0.7%; average about 0.2%. Albuminous contents gradually increasing in the course of the entire process.	Average about 0.4%.	Varying, usually considerably increased.	Normal.	Very considerably increased (up to 1%).	
Sugar contents of fluid (Worm-Müller's test).	Sugar always demonstrable.	Sugar tests nearly always negative, even after previous elimination of albumin.					
Bacterial contents of fluid.		In 30-50% of the cases tubercle bacilli demonstrable.	In the majority of the cases tubercle bacilli; post mortem always.	Intracellular diplococci; meningococcus of Weichselbaum and kindred types.	Pneumococci, staphylococci, and streptococci; typhoid, influenza, and colon bacilli.		
Special characteristic findings. ⁴		Coagulation of clear fluid, lymphocytosis, excessive pressure, and tubercle bacilli.	Varying findings in repeated punctures; pus cells and meningococci.		Creamy pus with various pathogenic germs.		

¹ All methods of measuring the pressure, in which before or during the procedure considerable quantities of fluid are drawn, are of no use (in children). By the use of mercury manometers this drawback can be avoided. The speed at which the fluid is voided is no criterion for the existing pressure.

² Technic of the process: The centrifuged sediment is treated exactly like blood in the preparation of slides. Fixation by alcohol-ether. Staining: triacid, azure, or eosin-haematoxylin.

OBTAINED FROM LUMBAR PUNCTURE

The assistant holds the anterior part of the pelvis with both hands (not shown in the illustration) and simultaneously been cleansed, etc., the operator, standing at the patient's left side, palpates with his left forefinger the second to fourth and directly forward in the median line into the interarcual space, until the resistance opposing the progress of the needle (see illustration). The pressure during quiet respiration and at the medium phase of respiration is read off at the should not be materially reduced below normal. The wound is closed by adhesive plaster.

Serous acute meningitis.	Chronic internal hydrocephalus (acquired).	Cerebral tumor.	Meningismus (in infections, intoxications, and reflex irritation).	Functional neuroses (epilepsy in paroxysms, tetany, chorea).	Sinus thrombosis.	Other pathological conditions and generally suggestive symptoms.
Nearly always increased.	In progressive cases (stages) considerably increased to about 60 mm.	Behavior varying, sometimes considerable increase.	In the average, slightly increased, never excessive, maximum about 45 mm.	Usually increased.	Often increased.	Rapid sinking of the pressure after drawing a little fluid, with occlusion of communication to the cerebral region of dissemination of the fluid; pressure zero at "punctio sicca" (dry tap). ³ Normal or low pressure with good cardiac function excludes exudative affections.
Clear, colorless.	Nearly always perfectly clear.	Varying, usually clear, carrying sometimes blood, sometimes flakes.	Usually clear; on closer inspection with light falling through and under agitation, often finest specks recognizable.	Clear.	Usually tinted with blood; often brownish green.	Bloody at puncture (unchanged appearance of elements, gradual decrease of blood contents, in various portions during voiding, strong tendency to coagulation), in hæmorrhagic pachymeningitis, cerebral and meningeal hæmorrhages, basal fractures, in (birth) trauma (old blood discolored, corpuscles disintegrate, coagulability absent).
No coagulation.	No change.	Exceptionally coagulation in net form.	Sometimes occurrence of a sediment which is just perceptible; no coagulation.	No change.	Sedimentation of disintegrated blood masses; (old blood) no coagulation.	
Like normal, or lymphocytes, somewhat more abundant.	Like normal.	Frequently increased uninuclear and polynuclear cells; also tumor particles (?).	Slightly increased lymphocytes and rarely polynuclear leucocytes.	Mononuclear and polynuclear cells.	Slightly increased lymphocytes.	A few lymphocytes are also found in cerebral sclerosis, poliomyelitis, syphilitic meningitis, otitis interna, whooping-cough, etc.
$A \leq 0.1\%$.	Nearly always normal.	Often increased, exceptionally up to 0.8%.	Rarely slightly increased $A \leq 0.1\%$.		Often somewhat increased.	$A > 0.1\%$ only in exudations or tumors. $A > 0.5\%$ only in tuberculous and suppurative meningitis (aside from the cases with puncture hæmorrhage).
Sugar often not demonstrable.	Sugar always demonstrable.	Sugar usually demonstrable.	Sugar demonstrable.			Absence of sugar: usually exudation.
		With tubercles, possibly tubercle bacilli.	In infections various bacteria were found, without inflammatory reaction of the meninges.			Mixed infections not seldom in tuberculous and cerebrospinal meningitis. In atrophies often intestinal bacteria.
Increased pressure with clear fluid.	Increased pressure, with otherwise normal conditions.					

³ No fluid is voided ("punctio sicca" or dry tap) because technic is faulty; or because the foramen of Magendie is occluded by a tumor; also in hydrocephalus; in jelly-like exudate; in cerebral hypertrophy, etc.

⁴ The findings as to permeability of the meninges, specific gravity, toxicity, and osmotic pressure of the cerebrospinal fluid are of no particular practical importance. The quantity voided is no criterion for the quantity present.

182 DIFFUSE HYPERÆMIA OF THE SKIN

A. UNIVERSAL (OR NEARLY UNIVERSAL) DIFFUSE HYPERÆMIA OF THE SKIN.

1. Physiological erythema neonatorum (Appears in the first 2 days of life in children with no pathological signs; subsequent fine desquamation).
2. Exfoliative dermatitis I (Appears almost always between the eighth and twentieth days of life; commencing usually at the lower half of the face (from labial fissures), extending gradually over the whole body; epidermolysis; desquamation of the epidermis in large friable scales; seldom formation of vesicles on reddened ground).

B. MORE NARROWLY CONFINED DIFFUSE HYPERÆMIA OF THE SKIN.

1. *Superficial Hyperæmia Without Œdema or Pain:*

- (a) Erythema in fever and various acute infections: Influenza, typhoid, pneumonia, and others (Especially on the cheeks; increased lustre of the eyes. In pneumonia sometimes on one—the affected?—side).
- (b) Erythema in psychic excitation.
- (c) Erythema in intoxications, atropine, etc.
- (d) Erythema as true vasomotor neurosis.—Organic and functional nervous affections. “Taches cérébrales,” “raies meningitiques,” Trousseau’s spots in meningitis, measles, exophthalmic goitre, also perhaps in rachitis.

2. *Permanent Hyperæmia Without Œdema* (at the cheeks).—“Hectic” hyperæmia in pulmonary tuberculosis; mongoloid idiocy.3. *Hyperæmia with Other Inflammatory Manifestations; Œdema, tension, pain, and fever.*

Simulated by diffuse urticaria with facial œdema.

- (a) Erysipelas (Lustrous dark red color, circumvallate borders which gradually extend; usually originating from epidermic defects*.)
- (b) Infectious megalo-erythema (Active, sharply defined hyperæmia with insignificant symptoms of infiltration of the somewhat painfully tense mucous membrane of the cheeks; usually little change of temperature; body exhibits a roseolar eruption; contagious!).
- (c) Erythema caloricum, sun-burn, scalding and burning of the first degree (History! localization!).

*Erysipelas in umbilical infections frequently begins at the (uninjured) scrotum. In the newborn the otherwise characteristic erysipelatous wall is absent.

B. MORE NARROWLY CONFINED, etc.—Continued

- (d) Intertriginous erythema (Moist, shiny, painful hyperæmia at places of predilection), caused by irritating organic secretions; *e.g.*, intertrigo.
 - (a) On buttocks and genitals in (thrush-) diarrhœa of nurslings, where there is insufficient and irrational nursing;
 - (β) on skin of abdomen and thighs in ectopia vesicæ;
 - (r) in articular flexions in obesity, hyperidrosis, rachitis;
 - (δ) of the upper lip in rhinitis, etc.
 - (e) Erythema after applications to the skin: Sinapisms, mustard packs, hot moist turpentine stupes, etc.
 - (f) Erythema over inflammatory foci, along with skin eruptions (variola vaccina, etc.).
- 4. Hyperæmia with Chronic Infiltration of the Skin:**
- (a) Diffuse, hereditary syphilitic erythema (On the palms, soles, the fingers, toes, and buttocks, skin tougher (like buck-skin), dark, smooth, with metallic lustre, lacquered, as if with broad scales).
 - (b) Chilblains, erythromelalgia, and many others.

183. PUNCTATE OR SPOTTED HYPERÆMIA OF THE SKIN

Type I. ROSEOLA (Usually merely isolated, small, round, red, sometimes transitory specks, which disappear on pressure by the finger):

- (a) Typhoid roseola (Localized, typical, rather pale red specks, persisting for a few days, sometimes slightly papular, up to lentil size, round, usually sparse, between mamillary and umbilical levels; appearance at the beginning or in the middle of the second week of illness).—Abdominal typhoid.
- (b) Roseola with other infectious diseases (Often prodromal, preceding exanthematous eruption; always transitory), especially in varicella, variola vaccina, cerebrospinal meningitis, pneumonia, rheumatic polyarthritis, sepsis (also sepsis neonatorum), mumps, miliary tuberculosis, influenza, non-specific angina, whooping-cough, erysipelas, [diphtheria].
- (c) Roseola in intoxications, botulism, etc.
- (d) Roseola in skin irritations through compresses, etc. (*Folliculitis*).
- (e) Sudamina rubra (At regions of profuse perspiration).
- [(f) Roseola in cutaneous miliary tuberculosis.]

NOTE.—Fleabite efflorescences differ from rosela by the generally visible central aperture of puncture and by often causing a slight hæmorrhage; "purpura pulicosa."

Type II. MEASLES-LIKE ERUPTION (Irregularly and often indistinctly defined specks, at first only punctate, later growing to lentil size and beyond):

- (a) Measles (At first follicular, punctiform, then ragged, starry little spots, raised slightly or not at all, with a central follicular nodule, seldom a vesicle. Often blood effusions. Beginning at the hairy part of the head and in the face, slowly extending for 3 days).
- (b) German measles (Measles-like spots, not papular, slight desquamation, lentil size, roundish, expansive, seldom confluent, at first usually with a central nodule, later always without it. No enanthem before the exanthem; scalp, palmar, and plantar surfaces also involved, on the extremities especially the extensor surfaces. Exanthem lasting only 2 or 3 days at the most; usually no fever, never diazo reaction).
- (c) Infectious megalo-erythema (on the trunk) (Eruption rubeolar).
- (d) Exanthemata, accompanying other infections, especially diphtheria, varicella, variola vaccina (Often distinguishable from measles only through concomitant circumstances, course of the fever, etc.).
- (e) Eruptions following the use of medicines (antipyrin, chloral hydrate, etc.) and after the injection of heterogeneous sera (serum disease) (Usually 8 to 12 days after the injection, not universal, usually leaving the face free, frequently starting from the place of injection and confined to the same; otherwise symmetrical; often accompanied by high remittent fever and albuminuria. Has nearly always been preceded by a sudden urticarial eruption; in the further course it may sometimes be recognized that the exanthem belongs to Type IV mentioned below; no catarrhal manifestations).
- (f) Scarlet fever with an atypical course (*scarlatina variegata*) (Eruption in groups between pale portions of integument; face less attacked than trunk or entirely free. Exanthem appearing at an earlier period than in measles; vomiting and angina).
- (g) Urticaria with an atypical course.
- (h) Blush erythema (At the upper pectoral triangle of older girls).

Type III. SCARLET FEVER-LIKE ERUPTIONS (Mostly minute, intensely red, thickly scattered specks, often on diffuse red ground. Skin velvety; as if finely sprinkled with red ink):

Type III, etc.—Continued

- (a) Scarlet fever (The finest points somewhat raised, at first separated, later connected by the red base; exanthem spreading from neck and chest within a few hours over face and trunk, in 24 hours also over the extremities; the middle facial triangle always remains free, a few other parts of the body frequently so; on the cheeks complete confluence. Some itching. Typical fever course and concomitant manifestations).
- (b) "Fourth disease" (Dukes) (Distinguished from scarlet fever principally by the milder course, and the absence of manifestations of the mucous membranes and complications).
- (c) Exanthemata accompanied by other infectious diseases, especially typhoid, pneumonia, influenza, sepsis, diphtheria, varicella (The red dots which always precede scarlet fever and merge into the surrounding red coloration either late or not at all are absent).
- (d) Eruptions after the use of medicines (especially atropine, quinine, salicylic acid, opium, mercury) and injection of heterogeneous sera (serum disease) (Occurring between the second and twentieth, mostly however between the eighth and fourteenth day after the infection, scarlet fever-like, but less intensely red and more diffuse; localization like scarlet fever, but less intense; often beginning at the point of injection, causing not infrequently regionary enlargement of the glands, albuminuria, remittent fever; no vomiting; no desquamation).
- [(e) The so-called "metadiphtheritic scarlatinoid" (During convalescence from diphtheria, also in non-injected patients; similar to the scarlet fever-like serum exanthem, but accompanied by angina and permitting diazo reaction) is probably a true scarlet fever condition running a modified course on a soil immune to diphtheria.]

Type IV. ANNULAR ERYTHEMA (eruption in the shape of rings and arches). (Larger, somewhat raised spots, which, by becoming paler in the centre with a livid hue, assume an annular shape, and in the further course, by peripheral spreading, confluence, and partial disappearance, form figures in the shape of bays and landscapes).

- (a) Multiform exudative erythema (Typical localization on the extensor surfaces of the extremities, dorsal surfaces of hand and foot; usually febrile; no desquamation; eruption transitory; often articular pains, cedema, albuminuria as concomitant manifestations).

Type IV, etc.—Continued

- (b) Toxic eruptions after the use of medicines (quinine, salicylic acid, etc.), after serum injections and in the company of endogenous toxicosis in gastro-intestinal affections, disturbance of digestion.

Type V. SYPHILOID (Yellowish or brown-red, salmon-colored or copper-colored round spots, with a tendency to lift the epidermis; usually without distinctly recognizable exudation): Macular syphilides (Insinuating appearance, of long duration; border often circumvallate, surface dark, lustrous, scaly; frequently affecting the soles and palms.)

194. PIMPLES, NODULES, AND PAPULES

1. *Urticaria* (Pimples, suddenly appearing, transitory, flat, itching, usually pale in the middle):

- (a) Ectogenous intoxications: Santonin, quinine, salicylic acid, antipyrin, opium, balsams, iodine, and bromide.
 (b) Endogenous intoxications: Dyspepsia, gastro-intestinal catarrh, intestinal parasites, cholæmia.
 (c) Here also belongs the reaction to subcutaneous administration of heterogeneous serum: Serum disease (8 to 12 days after the injection, often confined to the point of injection, otherwise usually symmetrical. Other exanthemata follow).

[(d) Infectious diseases: Diphtheria, influenza, mumps.]

[(e) Neuritis.]

2. *Lichen Urticatus*, urticaria papulosa, strophulus infantum (Disseminated over the whole body, usually excluding the head, indurated, itching nodules, single or grouped, with a red areola, up to lentil size, often carrying a little scab or minute vesicle; breaking out rapidly in crops, soon to disappear again spontaneously, often by the side of pimples and "urticaria factitia"; slight glandular swellings).—Same causes as in urticaria (acting on the soil of a degenerative diathesis).

3. *Atypical Varicella* (*varicella acuminata*).

[4. *Skin Lymphoma* in chronic lymphatic leukæmia.]

5. *Papulous Syphilide*.

6. *Rheumatic Nodules* (Subcutaneous painful nodules under the unchanged skin, varying from small to lentil size, occurring in the course of acute polyarthritis rapidly and especially situated near the articulations).

7. *Milium* (especially in the (immature) newborn at the nose, forehead, etc.), comedones, eczema, prurigo, scrofulous lichen, lupus, scabies, acne, etc.

185. FORMATION OF VESICLES AND CYSTS

1. *Vesicles with (almost) Quite Clear Contents:*

- (a) Varicella (Irregular dissemination over the entire body; hyperæmia absent or circumscribed, by the side of the pale vesicles, with which at times it greatly interferes; preceding papular stage either very short or absent; no prodromes).
- (b) Miliaria crystallina (Uniform transparent, minute vesicles (contents with acid reaction) upon quite normal skin, especially anteriorly at neck and trunk, itching!).—Hyperidrosis.
- (c) Scarlatina miliaris.
- (d) Lichen urticatus.
- (e) Eczema.

2. *Vesicles with Purulent Contents:*

- (a) Herpes (Always grouped, at certain places of predilection, accompanying infectious diseases).—"Febris hepatica," pneumonia, cerebrospinal meningitis, influenza, diphtheria, malaria, infectious gastritis, pyæmia, miliary tuberculosis, scarlet fever, [measles, typhoid, mumps].
- (b) Herpes zoster and herpes in neuralgia, neuritis, and myelitis (Outlined by the course and expansion of a sensitive cutaneous nerve; associated pain in children usually not very severe).
- (c) Variola vaccina.
- (d) Septic cystic eruption in the newborn and in nurslings (Sudden appearance of purulent cysts (frequently also hæmorrhagic) disseminated over the entire body, up to the size of a pea and soon changing into ulcers).—Septicæmia, especially after intestinal affections (bacillus pyocyaneus and others). Umbilical sepsis.
- (e) Impetigo, ecthyma, dermatitis herpetiformis.

3. **LARGE, ISOLATED, FLACID, UNILOCULAR CYSTS:**

- (a) Benign pemphigus, predominating in newborn and young nurslings (Frequently without any other pathological manifestations, occurring in limited number, chiefly on the trunk and thighs, never on the palms or the plantar surface of the foot, on normal skin, distinct cysts up to the size of a hen's egg, with good healing tendency).
- (b) Malignant pemphigus (and pemphigus foliaceus) (Very numerous cysts of varying size on hyperæmic skin, confluent and rapidly reforming; eczematous moistening and exfoliation of the skin. Secondary infections, high fever).

3. LARGE, ISOLATED, FLACCID, UNILOCULAR CYSTS:—*Continued*

- (c) Syphilitic pemphigus (Mostly congenital, with flaccid and smaller cysts on the palms and the plantar surfaces of the foot, appearing at the latest during the first week of life; cysts but slightly filled, contents very cloudy, often purulent and bloody).
- [(d) Exfoliative dermatitis II (After preceding diffuse hyperæmia (*see No. 182*) and epidermolysis; along with folded, furrowed skin is vesicular detachment, which later on leads to ulcerative processes).]
- (e) Pemphigus in neuritis, neuralgia, etc.
- (f) Pemphigoid measles.
- (g) Bulbous erysipelas.
- (h) Burns and freezing, "second degree."

186. NECROTIC, ABSCESS-FORMING, AND ULCERATIVE CONDITIONS OF THE SKIN

- 1. Decubitus after severe infectious diseases, in certain spinal affections.
- 2. Multiple folliculitis in infectious intestinal affections of nurslings.
- 3. Furunculosis after infectious diseases (typhoid, measles, erysipelas), in scrofulosis, in diabetes mellitus, chronic arthritis.
- 4. Abscesses of the skin, panaritium, ecthyma, and gangrene after infectious diseases, in neuritis, etc.
- 5. Tuberculous and syphilitic forms of affections.

187. DESQUAMATION OF THE SKIN

A. AFTER PRECEDING ERUPTION.

- 1. *Exfoliative Dermatitis and Pemphigus.*
- 2. *Physiological Desquamation of the Newborn.*
- 3. *After Acute, mostly Exanthematic Infections:*
 - (a) Scarlet fever (On the trunk, in small scales and platelets; on hand, foot, thigh, and nates in large lamellæ).
 - (b) Measles (Often insignificant, nearly always furfuraceous, exceptionally lamellar on the face; inner surfaces of hand and foot free; duration until fourteen days after the eruption).
 - (c) German measles (Slight and inconstant; if present, occurs by the third or fourth day after the eruption).
 - (d) Typhoid (Furfuraceous or lamellar, predominately on the trunk (very likely from miliary fever!) usually 10 to 15 days after the recession of the fever).
 - (e) Erysipelas, influenza.

A. AFTER PRECEDING ERUPTION.—*Continued*

(f) Syphilis (Large, lamellous desquamation with mirror-like lustre on hands and feet; furfuraceous, at the eyebrows and over older efflorescences of various kinds; scales often very coarse; squamous syphilitide).

4. *After Toxic and Other Exanthemata*; especially after mercurial medication.

B. WITHOUT PRECEDING ERUPTION.

1. Disturbance of nutrition of the skin in cachexias (atrophy, tuberculosis, diabetes mellitus) and in neuritis.
2. Congenital ichthyosis, psoriasis, herpes tonsurans, mycotic and squamous eczema, seborrhœa.

188. ABNORMAL DRYNESS OF THE SKIN

A. HABITUAL.

1. Cachectic conditions; Tuberculosis, syphilis, chronic affections of the liver.
2. Polyuria.—Diabetes mellitus and insipidus.
3. Myxœdema and other functional derangements of the thyroid gland.

B. ACCIDENTAL.

1. Formation of œdema; acute febrile infections during the rising of the fever.
2. Paralysis of the sympatheticus.

189. INCREASED PERSPIRATION

1. *Physiological Sweats* (Muscular work, psychic effects, heat) *from Pathological Causes*: Convulsions in tetanus, pseudotetanus, tetany, rabies, [epilepsy]; dyspnœa; hydropathic and medicinal therapy; violent sensory irritations.

2. *Universal Perspiration in (Rapid) Decline of Elevated Temperature*, especially in the crisis of infectious diseases (less frequently precritical or prodromal): Pneumonia, pyæmia, polyarthritis, cerebrospinal meningitis, malaria, tuberculosis (Here less frequently than in adults).

3. *Hyperidrosis at One or Both Halves of the Body or on Single Areas of the Integument*.—Often along with anidrosis of other parts of the body; occurrence also at places of the body which are otherwise free from perspiration. Spontaneous, "reflex" and "paradoxical" (on irritation by cold):

- (a) Cerebral affections: Hemiplegia, polienccephalitis, sinus thrombosis (Perspiration in the middle superior part of the face).

3. *Hyperidrosis*, etc.—*Continued*

- (b) Spinal affections: Myelitis, poliomyelitis, tabes.
- (c) Affections of the peripheral nerves, especially irritation of the sympathetic nerve: Neuritis, trauma.
(a to c frequently in paralyzed parts of the body.)
- (d) General neuropathy and functional neuroses: Hysteria, arthritism, migraine, exophthalmic goitre, epilepsy.
- (e) Reflex perspiration neuroses (Unilateral profuse perspiration in psychic affections, from heat, in masticating seasoned food, and "paradoxical" (on irritation by cold); frequently in the effective area of a certain sensitive nerve).
- (f) Constitutional anomalies: Rachitis (Especially perspiration of the head in sleepless restless children), infantile scurvy, affections of the thyroid gland.
- (g) General physical debility: Tuberculosis, anæmia, convalescence, agony, collapse.

190. **ŒDEMA OF THE SKIN WITHOUT ALBUMINURIA**(for œdema with albuminuria *see* No. 109)

A. ACTUALLY OR BECOMING MORE OR LESS UNIVERSAL; beginning usually at the eyelids or ankles.

Simulated by excess of fat, myxœdema, sclerema adiposum, emphysema of the skin.

Occurrence general: Hydræmic and toxæmic injury of heart or wall of vessels.

1. Cachectic conditions, congenital and acquired physical debility, athrepsia, sclerœdema neonatorum, congenital syphilis, tuberculosis; enteritis (Usually together with ascites; œdema, especially in the eyelids, dorsal surfaces of hand and foot, genitals; urine of high specific gravity).
2. Nearly all forms of infantile anæmia, especially acute leukæmia, pseudoleukæmia, splenic anæmia, also chlorosis.
3. All forms of hæmorrhagic diathesis.
4. Pathological group including erythema exsudativum multiforme and nodosum, and urticaria. Serum disease (Especially in the face).
5. Conditions of debilitated cardiac function: Congenital heart lesions; myocarditis, paroxysmal tachycardia ("Cardiac œdema," predominantly in the dependent and distal parts of the body).
6. Accompanied by acute infections, or after the same: Convalescence from scarlet fever (Usually in the third week). Measles, typhoid, polyarthritis (Superficial, pseudophleg-

A. ACTUALLY OR BECOMING MORE OR LESS UNIVERSAL, beginning usually at the eyelids or ankles.—*Continued*

monous œdema with erythema and pain), sepsis neonatorum, influenza, varicella, and other infectious diseases.

7. Intoxications of ectogenous origin: Salicylic acid, antipyrin.

8. Ischuria, dysuria, and functional insufficiency of the kidneys without degenerative or inflammatory changes (through retention of chlorides and phosphates).

[Under exceptional circumstances, albuminuria is permanently absent also in acute nephritis; also in diphtheritic nephritis ("Renal œdema," often superficial, usually noticed first in the eyelids).]

9. Diabetes mellitus.

10. Constitutional neuropathy: Arthritism, hysteria, exophthalmic goitre; also special causative factors; *e.g.*:

(a) Irritations, wet and cold (*see B*, 4); here possibly together with paroxysmal hæmoglobinuria.

(b) Excessive salt in food (especially in infants).

B. LOCALIZED.

1. *Local Disturbances of Circulation*: Obstruction of the efferent venous flow. Thrombophlebitis in acute infectious diseases, chlorosis, cachexia, sinus thrombosis; *i.e.*:

(a) Longitudinal sinus: Bilateral œdema of the face.

(b) Cavernous sinus: Unilateral œdema at the lid or of the face.

(c) Transverse sinus: Œdema behind one ear at mastoid process and nucha.

(d) Pressure on the abdominal veins (tumors, ascites, peritonitis): Œdema of the lower extremities.

(e) Pressure on the superior vena cava (enlargement of bronchial glands): Œdema of the face and upper extremity (Especially on the right side, intermittent).

(f) Increased pressure in the superior vena cava (whooping-cough and other cough affections: (Œdema of the face, "facies pertussea.")

(g) Extensive meteorism: Œdema of the abdominal integument.

2. *In the Neighborhood of Inflammatory Foci*, "collaterally":

(a) Of the eye: Conjunctivitis (*e.g.*, in measles), blennorrhœa.

(b) Of the face, unilaterally: Mumps (Often the entire half of the face, simultaneously the characteristic

B. LOCALIZED.—Continued

harder tumor in the fossa in front of the lobule of the ear). Alveolar abscess, periostitis from carious teeth, noma, erysipelas (Pale, with a waxy lustre).

- (c) Under the chin: Angina Ludovici, periostitis.
- (d) On the thoracic wall: Pleural empyema.
- (e) In the groin: Appendicitis, abscess of the psoas.
- (f) About the joints: Polyarthritides, osseous and muscular affections.
- (g) Also common in affections of the skin, accompanying infected wounds, in snake-bites, etc.

3. *Cerebrospinal and Peripheral Paralysis and Spasm*.—Transverse myelitis, poliomyelitis, tetany, neuritis (also without paralysis).

4. *Effect of Cold and Wet* on the healthy, and especially in individuals with an angioneurotic tendency (Superficially in fingers and cheeks, often symmetrical). Here also belongs submental induration from frost-bite (*see No. 191*).

191. INDURATION OF THE SKIN OR SUBCUTANEOUS TISSUE

(especially in the newborn)

Simulated by muscle rigidity (*e.g.*, in the face in trismus neonatorum), by elastic hard œdema.

1. Sclerema adiposum (Skin not pliable; immovable, rigid, parchment-like, dry, without lustre, whitish, waxy consistency, does not pit on pressure; arrest of movement; face involved, interference with suckling. Penis and scrotum free. Decrease in volume of the parts involved, subnormal temperature).—Physical debility, disorders of nutrition, loss of water, coldness of the newborn as well as of very ill older nurslings. [Exophthalmic goitre.]

2. Sclerœdema neonatorum (Skin doughy, plump-elastic, cool, but pliable to the touch; glistening, palely cyanotic or with yellow spots; consistency softer; digital impression remains; involving chiefly face and lower half of body, including the external genitals. Increase in volume of affected parts, subnormal temperature).—Disturbances of circulation of all kinds, in the newborn [seldom in the first few weeks of life]. Occasionally combined with adipose sclerema.

[3. Scleroderma (and atrophy of the skin) (Chronic course, no decrease of temperature, skin rigid, parchment-like).]

4. Submental induration of cellular tissue from freezing (In anæmic children after staying out-of-doors in winter, or the application of an ice-bag; painful, rather indurated, median infiltrate, about the size of a plum, spontaneously disappearing after 1 or 2 weeks).

5. *Phlegmasia alba dolens neonatorum* (Similar to sclerœdema, but only unilaterally in one leg, and very painful).—Thrombosis of the femoral vein.

6. *Erysipelas neonatorum* (Skin hot to the touch, strongly hyperæmic, fever).

[7. *Acromegaly* (At the distal parts of the body).]

192. ITCHING OF THE SKIN

A. GENERAL, HABITUAL.

1. Neurosis of skin in disturbances of nutrition (anæmia, rachitis), and neuropathy.
2. Diabetes mellitus.
3. Intestinal parasites (especially nose! ?).
4. Certain forms of dermatosis: Hebra's prurigo (Skin tough, dry, pigmented, especially at the anterior surfaces of the legs, extensor surfaces of the arms and the crural region), eczema.

B. OCCASIONAL AND USUALLY LOCALIZED.

1. Eruptive period of many acute infectious diseases: Measles, varicella, variola vaccina.
2. Dermatoses: Lichen urticatus, urticaria, scabies, eczema, cholæmia.
3. Affections of neighboring mucous membranes: Vaginal discharge, oxyuriasis, retained smegma.

193. ALOPECIA

A. TENDENCY OR CONDITION CONGENITAL.—Hereditary syphilis, myxœdema, cretinism (Hair of the head sparse, brittle, without lustre, dry).

B. ACQUIRED.

1. Skin affections with local effects: Seborrhœa in syphilis (Especially at the eyebrows and eyelids), herpes tonsurans, eczema mycoticum, exfoliative dermatitis after erysipelas. Traumatic lesion in hydrocephalus (heaviness of the head) and in rachitis (rubbing of the occiput against the pillow).
2. Convalescence after severe infectious diseases; especially typhoid.
3. Degenerative diathesis: Diabetes mellitus.
4. Chronic poisonings: Arsenic.

194. HYPERTRICHOSIS

A. GENERAL.

1. Premature infants (Lanugo).

A. GENERAL.—Continued

2. *Habitus tuberculosus* (Especially between the shoulder-blades and also on the extremities, forehead, and cheeks; long eye-lashes).

B. LOCALIZED.

1. *Hypertrichosis sacrolumbalis*; *spina bifida occulta*; hairy nævus.
2. Hairy forehead in myxœdema, etc.
3. Hairy back in older children suffering from rachitis.

195. CHANGES IN THE FINGER-NAILS

1. *Changes in the Nail Tissue*: Loosening, ridging, desquamation, brittleness.—Syphilis, myxœdema, cretinism, acute infectious diseases; vasomotor disturbances in nervous conditions.

2. Malformation of the Nails:

- (a) Formation of ridges and furrows; originating in the nail-fold, pushing to the edge.—At height of or during convalescence from acute exanthematous infectious diseases, especially scarlet fever (and measles), also typhoid.
- (b) *Onychogryphosis*.—Peripheral paralysis. Congenital cyanosis, obstinate cough affections (together with all kinds of drumstick fingers).

3. *Suppurative Processes of the Nail-bed*: *Paronychia* and *onychomycosis*.—Hereditary syphilis, scrofulosis, diabetes mellitus, convalescence from measles, accidental injuries; infectious diseases, especially stomatitis, rhinitis, etc.

4. *Shedding of the Nails*.—Several grave skin affections of an inflammatory and atrophying nature: Exfoliative dermatitis, diabetes mellitus, chronic arsenic poisoning.

196. PALLOR OF THE SKIN

A. TRANSITORY PALLOR, appearing occasionally and recurring frequently.

1. *Asphyxia pallida neonatorum*, in malformations of the thoracic organs, injury during birth with cerebral lesion.
2. Psychic excitement, violent sensory irritations, fainting; collapse; angiospasm in (toxic) neurosis, tuberculous meningitis, etc.
3. Masturbation, intestinal parasites.

B. (MORE OR LESS) PERSISTENT PALLOR, RAPIDLY APPEARING.

1. Hæmorrhages of all kinds.
2. Acute gastro-intestinal affections.
3. Toxicoinfectious myocarditis, diphtheria, etc. (Pale yellow waxy color).

C. PERSISTENT PALLOR, APPEARING GRADUALLY.

1. Primary disorders of the blood and of the blood-forming organs (*see Table following*); also, many kinds of hæmorrhagic diathesis.
2. Constitutional anomalies: Premature infants, rachitis, status lymphaticus, degenerative diathesis; here often in connection with irrational feeding régime (overfeeding, underfeeding, persistent milk diet, etc.).
3. Debility after long-continued illness, cachexia. [Infectious diseases taking a chronic course: Tuberculosis (often latent as "pseudochlorosis"), syphilis, malaria, rheumatic polyarthritis.]

Certain cardiac affections, especially adhesive pericarditis, acquired and congenital valvular insufficiency (frequently without murmurs!) (Here often without oligochromæmia!).

Intestinal parasites, especially anchylostomiasis.

Chronic disorders of digestion, especially dyspepsia of school children, with abdominal dilatation, intestinal atony, constipation; gastric ulcer, dysentery.

Chronic nephritis; here also belongs intermittent albuminuria.

Chronic intoxications: Lead.

4. Rapid growth at puberty with "growth hypertrophy" (and dilatation?) of the heart; cardiac neuroses, as school sickness (Headache, palpitation of heart, dyspnœa, murmurs of relative insufficiency; diffuse impulse of the thoracic wall at cardiac beat; first sound augmented, sometimes doubled; no galloping rhythm; narrow thorax; other neuropathic signs).
5. Masturbation.
6. Chronic intoxications (phenol derivatives, certain metals).

197. CYANOSIS

Nearly all conditions which lead to dyspnœa (*see No. 74*); here only those cases are considered in which cyanosis is especially striking or where the other manifestations are less prominent.

A. UNIVERSAL CYANOSIS.

1. Many cases of stenosis of the upper air tracts and obstruction of many bronchial lumina.—Pharyngeal abscess, laryngospasm, croup, whooping-cough (paroxysm), mediastinal tumor, bronchitis, and bronchiolitis.
2. Pulmonary affections: Atelectasis, pulmonary apoplexy in the newborn, croupous pneumonia in little children, miliary tuberculosis, infiltration of various kinds. Diaphragmatic hernia.

196. C., 1, PALLOR OF THE

(In order to judge the degree of anemia present, the color of the skin of the exterior ear (not of the cheek) is findings are merely approximate and without

	According to age.	BLOOD FINDINGS.					
		Erythrocytes.		Hæmoglobin.		Leucocytes per c.c. of blood, in thousands.	Occurrence of especially characteristic elementary constituents.
		Per c.mm. blood, in millions.	Per 1 leucocyte.	Normal percentage in adults.	Per 1 leucocyte in normal percentage.		
Simple secondary and symptomatic anemia (comp. No. 196, B. and C.).	At every age.	— to about 2.	— to about 100.	— to about 25.	— to about 60.	(+) to about 20.	Only in younger children, poikilocytes.
Chlorosis	Only after the eighth year; chiefly in girls.	+ — (—) 3-5½.	—	— to about 50.	— to about 40.	=	Usually none.
[Progressive pernicious anemia.]	Over 1 year of age.	—! to about ½.	— to about 70.	—! to about 5.	+ = 100-200.	— to about 2½.	Poikilocytes (megalo- cytes and micro- cytes), megaloblasts, polychromatophilia.
Acute and chronic leukæmia ("lymphatic" and "myelogenous").	Beyond the suckling age.	— to about 1½.	—! to 1.	— to about 30.	=	+! to about 70 (and more).	Atypical forms of various kinds; lymphocytes with large vesicular nuclei, normoblasts. Cells with karyolysis.
Pseudoleukæmia (Hodgkin).	Beyond the suckling age.	— to about 1.	— to about 60.	— to about 25.	— to about 60.	(+) = to about 30.	None.
Pseudoleukæmic infantile anemia (splenic anemia; related blood picture; hereditary syphilis).	Only between the [3d] 6th and 24th months of life.	— to about 2.	— to about 80.	— to about 30.	(—) =	+ to about 50.	Abundant normoblasts, with figures of nuclear division, giantoblasts, poikilocytes.
[Banti's disease.]	(Very rare) in older children.	—	—	—	—	—	—
Physiological limits.	Newborn	5-8.	250-450.	100-140.	—	17-35.	Normoblasts (sparse).
	Sucklings	4½-5.	360-450.	70-95.	—	10-14.	Large lymphocytes; very sparse normoblasts and myelocytes.
	Older children ..	4½-5½.	450-600.	70-100.	—	7-10.	—

As compared to normal. { + means increased, — means diminished, = means little changed. (+) means slightly increased, (—) means slightly diminished. +! means considerably increased, —! means considerably diminished.

SKIN IN BLOOD DISEASES

to be examined, also the color of the mucous membranes of the lips and eyelids. The figures given under blood regard to extreme and exceptional cases).

Occurrence of hæmorrhages as complications.	Color of epidermis, occurrence of œdema.	Behavior of the spleen (and the lymphatic glands).	Other signs of the affection and concomitant manifestations.
No hæmorrhages.	Skin greenish gray in younger children, parchment color in older ones; seldom œdema.	Not infrequently enlarged spleen.	Emaciation, anorexia, constipation.
No hæmorrhages (outside of irregular menstruation).	Skin white to yellowish green; great pallor, setting in suddenly; seldom œdema.	No enlarged spleen.	Considerable physical debility, fainting; change of disposition; superficial respiration, cardialgia, pica; venous murmurs; vasomotor disturbances; polyuria.
Often severe hæmorrhages into the skin, from mucous membranes, and in the retina.	Skin white, wax-like, with yellowish tint; later also more pronounced pigmentation of the skin. (Edema occurs.	Splenic tumor only small or entirely lacking (if present, increasing toward the end).	Rapidly proceeding to grave general condition; physical debility and tendency to fainting. Urobilinuria, indicanuria. Relation to intestinal parasites and syphilis. Not infrequently termination in leukemia.
Often severe hæmorrhages, especially also in the eye (retina, vitreous body).	Skin often dirty yellow, earthy; at other times nearly normally red. (Edema.	Often enormously enlarged spleen, indurated and notched, enlargement of the glands.	Course varying according to character, dyspnoea, œdema, and exulceration of lingual follicles. Lymphoma formation of the skin. Dilatation of veins.
Hæmorrhages occur.	Skin often of extreme pallor.	Considerable enlargement of the spleen and especially the glands, notably the cervical and tracheal.	Course and manifestations similar to those of lymphatic leukemia. Often compression of the trachea and bronchi by glandular tumors; skin lymphoma. Fever.
No hæmorrhages.	Skin waxy, yellowish to meerschaum color. Light œdema.	Enormous indurated spleen, notched, mobile; no considerable glandular tumors.	Course protracted, but often favorable. Liver slightly enlarged, abdomen distended. Relation to rachitis and syphilis.
	Dropsy.	Enlarged spleen.	Icterus, urobilinuria, enlargement of liver.

A. UNIVERSAL CYANOSIS.—*Continued*

3. Paralysis and spasm of the respiratory muscles.—Trismus and tetanus, epilepsy, postdiphtheritic paresis.
4. Cardiac affections with engorgement of the pulmonary circulation: Congenital and acquired heart disease. Myocarditis, myodegeneration, pericarditis.
5. Changes in the consistency of the blood in infectious and toxic processes.—Pernicious icterus (Winckel's disease) in the newborn; other forms of sepsis neonatorum, scarlet fever, diphtheria, pneumonia, effect of specific blood poisons.
6. Cerebrospinal affections: Traumatic hæmorrhages into the meninges of the newborn. Asphyxia livida neonatorum.
7. Intoxications; *e.g.*, alcohol.

B. CIRCUMSCRIBED, LOCAL CYANOSIS.

1. Scattered, especially in (mechanically) impeded venous return flow.
2. Of the face: Sinus thrombosis, enlargements of the mediastinal glands. Tuberculosis of the bronchial lymph-nodes; heat-stroke.
3. Of the extremities: Venous thrombosis; erythema nodosum, pernio, erythromelalgia, Raynaud's disease, sclerœdema neonatorum.
4. In paralyzed parts of the body.

198. ICTERUS OF THE NEWBORN

A. WITHOUT PERCEPTIBLE DISTURBANCE OF THE GENERAL WELL-BEING, spontaneous recovery.

1. Idiopathic icterus neonatorum ("physiological") (Appearing 2–4 days after birth, lasting till the tenth to the twentieth day, subsiding spontaneously; color, lemon yellow with a greenish tint; general condition undisturbed, except a slight alteration of the weight curve; stools unchanged; urine shows undissolved pigment or none at all; connective tissue and sclera discolored not at all or not until late; discoloration begins at face and chest).

[2. Icterus in cephalic hæmatoma; blood resorption!]

B. WITH GRAVE GENERAL CONDITION AND FATAL COURSE, under manifestations of hæmorrhagic diathesis.

- [1. *Bronze Icterus with Hæmaturia* ("cyanosis afebrilis ictERICA pernicioSA with hæmoglobinuria," Winckel's disease) (Appearance first or second week of life, duration until death, which usually occurs after 3 or 4 days. Convul-

B. WITH GRAVE GENERAL CONDITION, etc.—Continued

- sions and progressive decline; color, brass or light bronze; darkened by cyanotic streaks; dark sediment in the urine (hæmaturia—hæmoglobinuria?); epidemic occurrence).]
2. *Icterus Due to Mechanical Obstruction of Bile:*
- (a) Congenital cholelithiasis, congenital obliteration, atresia of the bile duct (Congenital, or occurring soon after birth; death in a few weeks; skin colored dark yellow-green; stools completely acholic; urine shows considerable dissolved biliary pigment. Ascites; other malformations!).
 - (b) Catarrhal icterus of the newborn (Behavior as in catarrhal icterus of older children).]
3. *Icterus in Sepsis Neonatorum* (Frequently with umbilical phlebitis and acute fatty degeneration of the liver (Buhl's disease); occurrence varying, often after the first week; death after from 3 to 12 days; highly febrile septicæmia with intestinal manifestations; color, lemon yellow plus cyanotic, therefore greenish; stools contain bile; urine generally contains biliary pigment.)
4. *Icterus in Congenital Syphilis of the Liver* (Occurring shortly after birth, death in a short time under manifestations of hæmorrhagic diathesis; enlarged spleen; color, citron yellow on gray base; stools contain bile; urine contains biliary pigment).
- [5. *Icterus in Congenital Cirrhosis of the Liver* (Occurring immediately after birth, death in the first month of life, enlarged spleen; color, lemon yellow; stools contain bile; urine usually contains biliary pigment).]

199. ICTERUS IN OLDER CHILDREN

A. Biliary pigment and biliary acid in the urine; stools acholic, very fatty, stinking; syndrome of cholæmic autointoxication: apathy, bradycardia, pruritus, albuminuria and cylindruria, xanthopsia, hæmorrhagic diathesis. Liver usually enlarged, somewhat tender on pressure. Fever lacking or only slight.

1. Catarrhal icterus (Slight disturbance of digestion, with fever which usually precedes by a few days; course favorable, duration 1 to 3 (up to 6) weeks; no enlarged spleen; not infrequently epidemic).
2. Icterus due to other mechanical biliary obstruction:
Compression of the bile duct by œdema of the lymphatic glands, fecal masses.
[Compression of the bile duct by pancreatic affection.

A. Biliary pigment and biliary acid, etc.—*Continued*

Compression of the bile duct by renal, omental, and other tissues.

Obstruction of the bile duct by biliary calculi.

Obstruction of the bile duct through ascarides.]

B. Generally biliary pigment in the urine, stools only partly and periodically acholic; often alimentary glycosuria and other signs of functional hepatic insufficiency; liver generally enlarged, painful on pressure, icterus in primary and secondary affections of the hepatic parenchyma. (For differentiation, *see also Hepatic Tumor*, No. 59.)

1. Active hyperæmia (Subicterus, liver slightly enlarged, sensitive).
2. Congested liver (Slight icterus, with cyanosis; alimentary glycosuria; liver sometimes considerably enlarged).
3. Fatty degeneration (Liver may reach very large proportions; borders sharp. Substance indurated, seldom pain on pressure; icterus a late symptom).
- [4. Amyloid degeneration (Seldom with icterus).]
5. Abscess.
- [6. Biliary and alcoholic cirrhosis.]
- [7. Acute yellow atrophy in syphilis and in cases of doubtful poisoning. (Diminution of the hepatic volume after previous enlargement, appearance of characteristic urinary elements, afebrile course, severe toxic general manifestations, and hæmorrhages. Death).]

ADD to this: Icterus in acute and chronic infectious diseases:

- [1. In Weil's disease (Manifestations of an acute general infection; hepatic and splenic tumor, hemorrhagic diathesis, cyclical course, frequently with relapses).]
2. In the early stage of croupous pneumonia, influenza, diphtheria, scarlet fever, [typhoid,] *see B*, 1.
3. In the course of typhoid, measles, diphtheria, scarlet fever, pyæmia, sepsis, malaria, ulcerous endocarditis, syphilis, tuberculosis, *see B*, 3. Icterus in heart and lung diseases. Icterus in poisonings. Phosphorus, arsenic, iodoform, chloroform, alcohol, ether, chlorate of potash, morphine, male fern, santonin, tuberculin, fungus poisons, *see B*, 3.]

Icterus with hæmoglobinæmia and hæmoglobinuria (*see No.* 105).

200. HÆMORRHAGES INTO THE SKIN

Round or longitudinal spots or elevations, sometimes slightly vesicular, which do not disappear on pressure, which show a typical change of color, and which are often localized at the hair follicles.

A. HÆMORRHAGES INTO THE HEALTHY SKIN*, quite spontaneous or following upon some small traumatic injury.

1. Genuine hæmorrhagic diathesis and primary blood diseases: Purpura simplex (Almost without any other manifestations). Purpura fulminans (Very extensive, symmetrical, skin hæmorrhages on the extremities, rapidly developing and leading to death. Mucous membranes free). Purpura hæmorrhagica (With hæmorrhages of the mucous membrane). Peliosis (With articular affections). Infantile scurvy (More or less distinct bluish discoloration over the affected bones; but also purple spots on injured places). Hæmophilia, leukæmia, pseudoleukæmia, splenic anæmia, pernicious anæmia.
2. Septic and pyæmic processes. Independently or in the course of specific infectious diseases: Scarlet fever, diphtheria, influenza, typhoid, pneumonia, erysipelas, varicella, septic variola, malaria, endocarditis ulcerosa. Here possibly also belong cachectic septic processes after measles, taking a chronic course, in athrepsia of infants, in tuberculosis with cavity (mixed infections!).
3. Hereditary syphilis, especially when the liver is involved.
4. Toxic processes: Phosphorus poisoning, poisoning by iodine, arsenic, mercury, antipyrin, chloral, quinine, balsams, meat poison. Here also belongs serum disease (In stripes and weals, especially in cachectic individuals).
5. Increase of the blood pressure.—Cardiac hypertrophy in contracted kidney; violent movements of the body; extreme venous engorgement in vomiting, coughs, and spastic paroxysms.
6. Traumatisms of various kinds: In the newborn, birth trauma (Hæmorrhages on the chest and back); in older children, ill-treatment; chorea minor. Flea-bites (purpura pulicosa).
7. Organic cerebral affections and functional neuroses: Encephalitis, neuralgia, neuritis, hysteria.

B. HÆMORRHAGES INTO PREVIOUSLY EXISTING EFFLORESCENCES OF THE SKIN in the normal or abnormal course of exanthems: Measles, scarlet fever, roseola, varicella, lichen scrofulosus, insect bites.

NOTE.—Hæmorrhages into and under the skin in the newborn from the same causes as umbilical hæmorrhages (*see No. 63*).

201. PRONOUNCED SPOTTY PIGMENTATION OF THE SKIN

Pigmentation of the linea alba below the umbilicus frequently in healthy girls (less frequently in boys) at puberty.

Simulated by dirt spots.

*Especially hæmorrhages into the skin; similar conditions lead to hæmorrhages under the skin.

A. AFTER PRECEDING EXANTHEM (especially syphilis, also erythema solare, urticaria pigmentosa, serum disease) AND HÆMORRHAGES.

B. WITHOUT ANY PARTICULAR PRECEDING CHANGE OF THE SKIN.

1. Cachexia, especially chronic peritoneal and pancreatic affections (Mulatto color). Malaria, pernicious anæmia, chronic septicæmia, cretinism.
2. Addison's disease (Dirty yellow to earth-colored, even black, diffuse or spotted, especially at uncovered parts of the body; nails free, a few mucous membranes involved).
3. Long-continued medication or occupation with arsenic, lead, silver, or their compounds.
4. Bronze icterus in the newborn.
- [5. Exophthalmic goitre.]
6. Pityriasis versicolor and other dermatoses.

202. RHEUMATOID PAINS

(also pains in the region of the joints without any other local signs)

Simulated by osseous pains (*e.g.*, in infantile scurvy), neuralgia, syphilitic pseudoparalysis.

A. OCCASIONAL.

1. Acute infectious diseases (also prodromal): Measles, scarlet fever (Especially articulations of hand and vertebræ; multiple commencement from the fifth to the twelfth day of illness), severe diphtheria, typhoid, mumps, cerebrospinal meningitis, influenza, sepsis neonatorum, poliomyelitis, dysentery, non-specific tonsillitis, ulcerous endocarditis.
2. First stage of all forms of exudative arthritis (*see No. 203*).
3. Hæmorrhagic diathesis, rheumatic peliosis (Especially knee and ankle joints), hæmophilia (Usually knee joint, later other joints; appearing first at 4–10 years of age; previous history, hereditary taint!).
4. Serum disease (Beginning usually 8–12 days after injection; especially in the metacarpo-phalangeal articulations, also hand and knee joints; pains violent without objective finding; salicylic acid without effect).
- [5. Spinal affections: Tabes.]

B. HABITUAL.—Pernicious anæmia, arthritism, chronic hepatic affections, hysteria (with or without contractures and paralysis, changeable).

203. ARTICULAR EFFUSIONS AND INFLAMMATIONS

Simulated by affections of the bones (osteomyelitis, infantile scurvy, vertebral caries).

A. ACUTE.

1. Acute polyarthritis (Usually beginning in lower extremities, always several articulations (often also vertebral articu-

A. ACUTE.—Continued

lations), irregular abrupt attacks, transitory process, high fever, intense pain, periarticular œdema, specific effect of salicylic acid.)

2. Purulent arthritis in acute infectious diseases (often mon-articular, usually becoming suppurative, especially in smaller children; considerable swelling and œdema, with relatively slight general manifestations): Pneumonia, cerebrospinal meningitis, scarlet fever, typhoid, erysipelas, influenza, dysentery, sepsis neonatorum, septicopyæmia in older children. [Measles, varicella, mumps, diphtheria.]

Gonorrhœa (Often monarticular, usually knee, less often carpus and tarsus, painful with suppurative periartthritis (in younger children!), course long-continued, but usually benign, sometimes abortive; salicylic acid without striking effect!).

3. Hæmarthrosis.—Hæmorrhagic diathesis, hæmophilia, peliosis rheumatica (Especially lower extremities, often without inflammatory reaction, relapsing, together with other hæmorrhages).
4. Hydrops articulorum.—General dropsy; trophoneurotic disturbance in neuritis and spinal processes, also "idiopathically intermittent "

B. CHRONIC.

1. Chronic polyarthritis after acute affections (usually progressing centrifugally), primary chronic (centripetal).
2. Tuberculous arthritis, fungus (usually with osseous affections and diseases of the soft parts, especially lower extremities).
3. Syphilitic arthritis, late hereditary syphilis (usually afebrile, bilaterally at the knees, soft parts free; with Hutchinson's triad).

204. BONE PAIN

1. *Constitutional Diseases*: Infantile scurvy (Swelling and sensitiveness to pressure at terminations of diaphyses, often symmetrical; examination of mouth and gums, anæmia, sinking in of sternum, radiogram!). Rachitis (Epiphyseal). Osteomalacia, myelogenous leukæmia (Especially in the long bones of the lower extremities and in the sternum!).

2. Chronic Infectious Diseases:

- (a) Syphilis: Parrot's paralysis (Fusiform distention of the long bones in the epiphyseal region, osteochondritis and loosening of epiphyses). Gummata of cranial bones, periostitis (Especially at the anterior part of the tibia, cranium, and sternum). Syphilitic phalangitis (Distention of the phalanges without involvement of the soft parts).

2. *Chronic Infectious Diseases:—Continued*

(b) *Tuberculosis*: Spondylitis (Rigidity of the spinal cord, pain, fever, cold abscess, deformity). Tuberculous meningitis (prodrome!).

3. *Acute Infectious Diseases*: Osteomyelitis, primary or after typhoid (Especially tibia, humerus, ribs, and vertebræ). Pneumonia, measles.

4. *Hepatic Cirrhosis with Osteo-arthritis*.

5. *Rapid Growth* (Tarsalgia, painful flat-foot, tibial pain; disappear after several days' rest in bed; possibly formation of exostoses).

6. *Tumors*: Femoral sarcoma, maxillary sarcoma.

205. **MUSCULAR PAIN**

(partly in connection with inflammation of the muscles)

1. *Overexertion and Trauma* (here possibly also tears of muscle fasciæ):

(a) Sternocleidomastoid muscle, after injuries at birth (Usually noticed at the age of 4–6 weeks, nearly always the right muscle, median part, anterior border; oval, indolent tumor, the size of a hazel-nut, with a bluish glimmer, at first painful, fluctuating, later harder; skin movable; shortening of the muscle remaining).

(b) Abdominal recti muscles after violent coughing.

(c) Various muscles of the body in spasms, after gymnastic exercises, etc. (In the muscles of the calves with painful spastic attacks at night).

2. *Rheumatic polymyositis* [and suppurative polymyositis].—Sepsis, scarlet fever, typhoid.

3. *Acute Infectious Diseases*: Scarlet fever, typhoid, tuberculosis, influenza; also, especially, cholera nostras.

4. *Neuritis*.

[5. *Myositis ossificans, trichinosis*.]

206. **MUSCULAR ATROPHY**

1. *All Flaccid, Spinal and Peripheral Forms of Paralysis* (Rapidly setting in and progressing, reaching high degrees, frequently also involving tendons, bones, and skin).

2. *Many Spastic, Cerebral Forms of Paralysis* (in consequence of disuse) (Atrophy setting in late, progressing slowly, never reaching high degrees).—Especially infantile cerebral paralysis.

3. *Idiopathic, Progressive Muscular Atrophy*:

(a) Spinoneural forms (Beginning at pelvic girdle, lower extremities, later arms, progressing centripetally, symmetric, with paralysis. Reflexes diminished or abolished; often fibrillary twitchings and R.D.; family occurrence).

3. *Idiopathic, Progressive Muscular Atrophy :—Continued*

(b) Myopathic forms (Beginning in the face, shoulder, legs, progressing centrifugally, symmetric; atrophy without actual paralysis; no fibrillary twitchings, reflex excitability diminished, no R.D., no disturbance of sensibility; often lordosis; family occurrence).

4. *Amyotrophic Lateral Sclerosis* (Spastic paralysis with atrophy, patellar reflex present).

5. *Hereditary Ataxia.*

6. *Prolonged, Painful Affections of Bones and Articulations:* Fungus, caries, osteomalacia, rachitis, chronic arthritis, etc.

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GENERAL PROPHYLAXIS IN DISEASES OF CHILDREN

BY

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THROUGH prophylactic measures every physician should endeavor to protect from danger and disease those intrusted to his care and should consider it his foremost duty to coöperate in the great questions of hygiene, not only in public life, but especially in the smaller circle of his activities as family adviser. The children's physician is called upon, in exceptional measure, to give advice to mothers in regard to the care of the child's health and to offer his knowledge and experience in its behalf, so that the innumerable dangers which threaten its health and life may be warded off, and an existing predisposition may early be combated and suppressed, thereby bringing about the conditions necessary for perfect health. By collating the numerous data gathered from the observation of the child in health and sickness, valuable indices for the prophylactic rules and measures may be obtained.

The slow growth and development of certain organs and systems in infancy and early childhood, especially the digestive apparatus and the osteal and nervous systems, which have not yet attained the adult type; and the fact that in the early years of life the skin and mucous membranes, because of their greater susceptibility to infections and poisons, are a fruitful field for the various injurious influences which are always at work, compel the physician to give these organs particular care and attention in his endeavor to preserve their normal function. In much the same way, great vigilance is demanded of the pediatricist during the mental and physical development of the child, in warding off the dangers to various organs (*e.g.* the heart and the vascular and nervous systems), due to their inability to meet the demands made upon them, as at the beginning of school life and puberty,—epochs when the damage to them would be serious and perhaps permanent. The children's physician must therefore exert the utmost endeavor to minimize the dangers attending the frailties of this early period, brought about by rapid development and the school.

All the hygienic measures which serve as guides to the preservation of health in adults may be applied in childhood, in a slightly differ-

ent manner, and, usually, with more care. The elements from which these general rules are derived depend upon the proper carrying out of principles of the utmost cleanliness (asepsis), particularly in the care and feeding of the child; the younger the individual, the greater is the rôle played by it. Plenty of sunlight and clean fresh air are necessary, with protection from extremes of temperature, particularly against the sudden and abrupt changes in temperature which are so prolific a source of catarrhal colds. And not of least importance among these general measures for health is the careful and rational nourishment of the child, which can be accomplished by utilizing our experience and our knowledge of physiological principles based upon the course of development of the child and observation of its metabolism. It should also be our duty, in similar fashion, to build up systematically the bodily strength and develop harmoniously the mind and intellect.

The manner in which general hygienic measures may be carried out depends very largely upon the environment in which the individual lives. The more favorable the material condition of the parents the more favorable is the hygienic condition of the child's life likely to be. Poverty and affluence play an extraordinarily important part, particularly in regard to the abode and food of the child. It is generally well known, and proved by statistics, that in the crowded quarters of the poor, where the living rooms are ill ventilated, the water supply is insufficient, and the food-stuffs are not beyond criticism, the infant mortality and the spread of epidemics and morbidity among children are much greater than in those quarters where the people are in better circumstances. Similarly, the care which is taken of the child, as well as the carrying out of the measures which make for its physical and mental growth, depend upon the circumstances of the parent or guardian.

The enormous importance of the housing of the poor, when considered in its relation to questions of health, makes it urgent to strive to the end that they may participate in the advances made in home hygiene and live in a manner better than heretofore, at a rental low enough to be within their reach. Above all, the dwellings of the poor should be dry, well ventilated, and have running water. They should be built only in sections free from miasma, upon dry, non-swampy ground.

The scarcity of small dwellings is due to the great lack of space. Often the whole family is crowded in a single room, which is frequently the bedroom, living room, and kitchen combined, where the air is damp from clothes hung up to dry. Such inferior dwelling places as these offer the most favorable conditions for infection and the spread of many diseases, owing to the impure air and the accumulation of dirt and germs of all kinds. Other ills which almost inevitably result from these condi-

tions are anæmia, rachitis, and tuberculosis. Here is an opportunity for the government, for coöperative societies, and, last but not least, for philanthropic bodies to engage in social work whose possibilities are boundless, to give to the workers and their families cheap and hygienic homes. When carried out upon broad lines, as has been done in some places, the results are excellent. The prophylactic measures instituted prevent, or diminish, just those conditions which are of such serious import, and so fatal in childhood. Every physician should do all in his power to arouse interest in the effort to improve the hygienic conditions of the dwellings of the poor, to whom the matter is of far greater importance than it is to those in better circumstances.

The following suggestions in regard to the housing of the child, can, however, be carried out in part only by the better class of families:

It is desirable that, from a very early age, children should have their own bedrooms and not sleep in the same apartments as the parents. The question of æsthetics, as well as of health, make this advisable. At the beginning of school life, if not earlier, it is desirable that a play room or work room be fitted up for the child. Both rooms should be as large and airy as possible, easily ventilated, and dry. If possible, southern exposure should be selected. In neither room should the temperature be kept too high, 18°–19° C. (64°–66° F.) during the colder months should suffice. In the sleeping rooms of older children the temperature should be 14°–15° C. (57°–59° F.) and for children under three years, 18°–19° C. (64°–66° F.). Unless the temperature outside is entirely too cold, the sleeping rooms should not be heated at all; certainly not where the children are over three years of age. For infants, the heating of the bedroom can hardly be dispensed with. Formerly, the heating of rooms was best accomplished by means of a tile stove, but this has been succeeded in most places, at least in cities, by steam heat. Owing to the greater dryness of the air with this method of heating, the necessity for ventilating the rooms is even greater than in former years, and this is especially the case with bedrooms, the windows of which should be kept open all day. It is necessary also to purify the air in the living rooms by opening the windows frequently, for shorter or longer periods. Beside proper ventilation, it is important also to keep the rooms as free from dust as possible by wiping up the floors with a damp cloth at least twice daily. Painted floors, or those covered by linoleum, are easily kept clean and are least likely to allow accumulation of dust.

It is essential that the rooms be furnished in the simplest manner. The walls should be painted with white or gray oil paint, so that they may be frequently washed and readily disinfected. Light, washable hangings or curtains should be used at the windows instead of heavy portières, so that the room will not be darkened. Linoleum should be

used in children's rooms instead of carpet. Upholstered furniture likewise is unsuitable for children's rooms; plain wooden tables and chairs should constitute the furnishing. The height of the seats should be regulated according to the size of the child, and for school-work they are to be recommended in preference to the individual writing desks ("Verstellbare Schulbank"), at which children are more likely to lose their normal carriage than to correct deformities.

The toys which accumulate in great quantities from year to year, and which, though no longer used as playthings, are still retained in loving memory of childhood through school days and even beyond puberty, should not be permitted to stand about the play room, but should be locked in chests or closets, because of their tendency to collect dust. The homes of the children, and particularly the work room, must be well lighted and should possess an excellent source of illumination. Electric lights in hanging lamps, with ground glass bulbs and green shades, are best for artificial illumination because they do not consume the air, they are least harmful to the eyes, and are least likely to cause fire. Where this is not to be obtained, the familiar oil or petroleum lamps are still to be preferred to the gas flame, as being less trying to the eyes; the oil or petroleum lamp, however, soon heats and vitiates the air. According to my experience, the children's workroom is not complete without facilities for washing, so as to maintain cleanliness among them. I consider a swinging horizontal bar suspended from the ceiling of the room, a valuable addition to the home, for after the child has been seated a long while at work, the need for exercise is imperative.

In the sleeping room, too, the furnishings should be of the simplest kind and easily kept clean; with a bed, a washstand, a wardrobe, and a chest of drawers the installation is complete. The bed should be so arranged as to prevent the child being rendered too soft. A horsehair mattress to lie on and a linen sheet and a woolen blanket for covering, are better than a soft mattress and a feather-bed for cover. During the first three months of life and during the winter, a light down coverlet may be used. Iron beds are most easily cleaned and are not so readily infested by vermin. For durability and elegance, the so-called English beds occupy first place.

A baby carriage, or a basket suitably covered, to prevent the entrance of too strong a light, usually serves as the infant's bed. According to our idea, the cradle, that show-piece of the nursery, should be discarded, for the mother is apt to rock the child constantly in her endeavor to produce sleep, and thus make use of a sedative which may be harmful to the nervous system. The infant should have its own bed, in order to obtain plenty of clean fresh air, and also to obviate the danger of being "overlain."

The proper care of the child's skin is absolutely essential to healthy development. Further on I will explain why I consider daily bathing of the infant a necessity from the day of birth. According to ancient and honored custom among us in Germany, the cleansing bath for the young "world-citizens" is administered daily only to the end of the first year. After that time, the physician is repeatedly asked whether a bath once or twice a week will not suffice, and he is often told that more frequent bathing must weaken the child. This false point of view is difficult to combat. I consider it extremely necessary that no time limit be placed upon the matter of bathing; and when it is at all possible the mother should continue the daily bath until it has become a fixed habit in the child, in the hope that this excellent practice of caring for the skin may be continued throughout life. I should also like to see the evening sponge bath, which every mother gives her infant, continued through childhood. This procedure is best carried out with tepid water at 20°-22° C. (68°-72° F.). I am not in favor of hardening the child by pouring cold water over it or by using the cold shower. Instead of this, quickly washing the entire body with a large sponge dipped in lukewarm water, or wrapping it in wet sheets for a short time, is useful for strengthening the skin and the muscular system. Further on I will take up in detail the care of the infant's body.

In addition to the hygiene of the skin by means of the bath, the care of the mouth and teeth are part of the child's toilet. It has been found from general experience that the cleansing of the baby's mouth by wiping produces superficial injuries of the mucous membrane which lead to local infections and may result in serious constitutional troubles. Even if the use of an aseptic nipple were not dangerous, I would still object to the general use of "comforters" for the purpose of quieting the baby. The chief method of preserving the teeth in later childhood is by a careful cleansing of the mouth, by regularly rinsing it every morning and after the principal meals. The teeth should be cleansed after each meal with a tooth-brush of medium stiffness. As mouth washes, $\frac{1}{4}$ to 1 per cent. thymol or menthol solutions (a few drops added to a glass of water) may be used; or

R	Thymolis.....	0.25.....	gr. iv
	Acid. benzoic.....	3.00.....	gr. xlv
	Tinct. ratanizæ.....	15.00.....	ʒ iii ℥ xlv
	Alcoholis.....	100.00.....	ʒ iii ʒ iiss
	Ol. menthæ.....	0.75.....	℥ xii

M. Sig.— $\frac{1}{4}$ teaspoonful in a glass of water.

As a tooth-powder :

R	Magnesii carbonatis.....	5.00.....	ʒ i gr. xv
	Cretæ albæ præcip.,		
	Sodii salicylatis.....	āā 15.00.....	ʒ iii gr. xlv
	Ol. menthæ piperitæ.....	gtt. ii.....	gtt. ii

M. fiat tooth-powder.

Or :

R	Magnesi carbonatis,		
	Sapon. med.	āi 10.00.....	ʒ iiss
	Pulv. oss. sepis.....	80.00.....	ʒ ii ʒ v
	Ol. menthæ piperitæ.....	gtt. ii.....	gtt. ii
	M. fiat tooth-powder.		

The careful preservation of the milk teeth is something of a guarantee for healthy permanent teeth. The extraction of a milk tooth should be permitted only when the root has become inflamed and is the seat of a periostitis. All the care of the teeth is not comprised in local measures, however. It is necessary to exclude from the diet all articles, which, either directly or indirectly, may prove harmful; hence, sweets, and food or drink which is either too hot or too cold, should be forbidden.

Consideration of the child's clothing should begin in infancy. It should be of such a character as to allow the utmost freedom of movement of the trunk and extremities and not to interfere by pressure with the development of muscles and bones. For this reason binding up the infant in swathing bands has very properly been abandoned. The underwear should be soft and dry, to prevent irritation of the delicate skin. The principle should be borne in mind that through childhood there must never be a hindrance to free movement, or pressure upon muscles, bones, or internal organs. The cut and style of the baby garments may be left to the seamstress.

For older children, generally speaking, light washable materials are preferable to dark woollen ones, as being more hygienic. Moreover, the clothing of the child should be suited to the season and the weather. Thus in summer, light weight, light colored materials should be chosen, with as much of the body (neck, shoulders, and arms) uncovered as possible, so that the air and sunlight may have free access to the skin. It is not advisable to dress the child too warmly in winter, lest free perspiration be induced while it is exercising in the open air, which so readily leads to colds. Healthy children do not require scarfs or mufflers around the neck. Broad-brimmed straw hats are the best protections against the intense rays of the sun.

Certain articles of clothing demand especial attention on the part of the physician, in order that particular deformities and injuries to organs may be prevented, and that correct position and carriage of the child may be maintained.

The younger the individual, the more readily are the feet affected by improper foot gear. Proper shoes are of prime importance. These should follow the form of the foot, should be broad enough to give plenty of room to each toe, and should have a low, broad heel to insure a firm hold for the foot. Inasmuch as taste and fashion make it possible to become accustomed to certain shapes, it is to be hoped that in time the

fashion-madness which finds its ideal in narrow, pointed shoes wherein the toes are crowded together, will finally capitulate to common sense, especially when the people are made aware, through repeated advice in matters hygienic, of the harm which may result from wearing improper shoes. But the shoes alone are not always to be blamed for causing deformities of the feet, with consequent disturbance of the functions of the lower extremities and peculiarities in gait, for an unsuitable stocking is capable of causing similar harm by interfering with the development of the toes. A separate pocket should be provided in the

stocking for the great toe, in order to prevent its displacement toward the median line of the foot.

FIG. 8.



Constricted chest from corset.

High collars or neck-pieces, especially when worn tight around the neck, are to be avoided, as they are likely to cut off or interfere with the circulation of the large superficial veins of the neck, through which the blood must return from the head. A number of unpleasant symptoms are apt to appear after a time, such as dizziness, headache, congestion of the head, and general irritability, which are without apparent cause and are very puzzling to the physician unless he has observed the offending neck-piece. The round garter encircling the upper part of the leg acts, in a similar manner, as a tourniquet for the veins of the lower extremities, and, when worn from early childhood, may produce varicose veins and promote the for-

mation of ulcers of the foot.

The corset is the greatest and most serious menace to the health of the female sex. Owing to the dictates of fashion, which declare a waist beautiful only when encircled by a corset as by an armor, there develop after a time those injuries and anomalies of which the constricted chest (*Schnürthorax*) is characteristic. Here, above all, are to be considered the effects of pressure upon the abdominal organs, particularly upon the liver, and the displacement of the abdominal organs (*gastrop-tosis*, *enteroptosis*), with all their remote sequelæ, such as dilatation of the digestive organs, chlorosis in all grades, poor development, and nervous symptoms of all kinds. The earlier tight lacing is practiced and the more the shape of the thorax is removed from the normal, the more certain is the prolapse of the stomach, the less the opportunity for the blood-making system to escape disturbance, and the more persistent and difficult to cure are the consequent symptoms of disease. Children who do not wear corsets but who wear skirt bands which are too tight may

develop manifestations similar to those which occur from wearing tight corsets. It is part of general prophylaxis to preserve the natural form of the thorax in the young and to guard them from the evils induced

FIG. 9a.



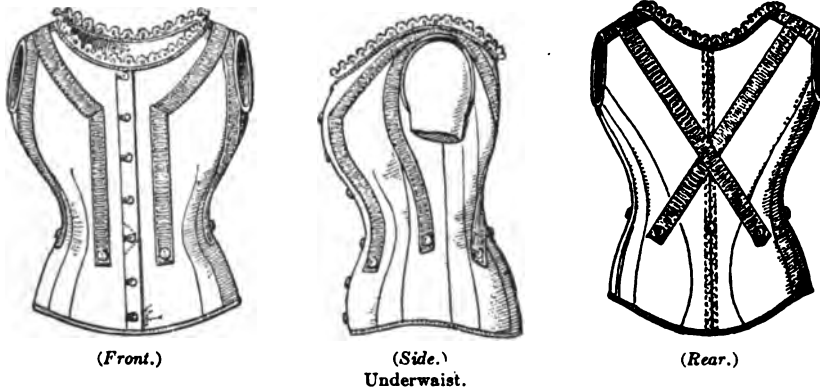
FIG. 9b.



A case of gastropotosis (enteroptosis) in a child of eleven years. Cure after three months by a reform in dress, and lung exercises.

- (a).—January, 1892. Corset since third year; anæmic, dyspeptic, hysteroneurasthenic. Distention of stomach with tartaric acid 3.00, sodium bicarbonate 4.00.
 (b).—April, 1892. A robust figure. Large increase in weight. Stomach and nervous symptoms have permanently disappeared.

FIG. 10.



by the wearing of corsets. But in order to get along without a corset, which lends support to the entire upper part of the female figure, including breasts and back, it is necessary that the girl's body should be strengthened by good nourishment, plenty of fresh air, and particu-

larly by exercise, such as gymnastics, tennis, rowing, etc., so that the muscles may be strong enough to furnish the requisite support without artificial aid. This end is fully attained, in my experience, when the development of the muscles of the chest and back is begun early. Instead of corsets, the girls ought to wear underwaists which have buttons to which the skirt and drawers can be fastened, thus dividing the weight of the clothes between the hips and shoulders. This manner of dress does not permit the wearing of heavy underclothing. The same result has also been attained by the so-called "reform" clothing, which often replaces the skirt by trousers. Whenever this kind of clothing is used and it is found that there is pressure upon the two floating ribs (which in women is particularly harmful), it should be made clear that the band of the skirt must be worn loosely and that the garment itself should rest snugly upon, and take its support from, the crests of the ilia.

After these general propositions, which include the whole subject of hygiene, it is perhaps best to consider the subject of prophylaxis as it is practiced during the various periods of childhood. The subsequent treatment of the subject is made with a view to the various phases of development at different periods of life. Depending upon the age, there occurs an occasional asymmetry or other defect, which will interfere with the healthy progress of the individual. This is sometimes produced by internal causes (as backwardness or sensitiveness of individual organs), and sometimes by external causes (as great bodily or mental strain).

The most important hygienic measure pertaining to the "epoch of the newborn," by which we understand the period from the moment of birth until the falling off of the cord and the healing of the umbilicus, is strict and thorough asepsis. Careful consideration of the process of involution of the vessels and the healing of the umbilical cord will make it apparent how, in the first weeks following the birth of the child, there are rich opportunities for infection of the umbilical wound with production of localized disease, or how the severest general infections may ensue through the entrance into the blood current of malignant organisms. It should also be borne in mind that not only is the umbilicus a possible point of entrance for infection during the early days of life, but that the delicate skin and easily injured, or previously injured, mucous membranes are a sensitive and receptive ground for the growth of pathogenic bacteria, and that the germs, which are normally present upon them may become active and multiply. Painsstaking care of the navel, therefore, until after the cord has fallen, is one of the most important prophylactic measures for the newborn. Bearing this in mind, the tying off of the cord should be performed under the most careful aseptic precautions, as soon as the pulsations in the umbilical vessels have ceased. After sterilizing the hands, the cord is to be tied with sterile

tape about two fingers' breadth from the umbilicus, and again at about the same distance toward the placenta, and divided between the two. Then, after the infant is bathed, the first knot is to be tightened and a second final knot is to be made. The ends of the tape are then properly arranged upon the abdominal wall, covered with dry sterile gauze, and fixed in place by the abdominal binder. This dressing is to be renewed in the same manner each day after the bath. In place of the usually unsatisfactory abdominal binder, one can, with advantage, use the Flick apron-bandage by which soiling through the urine and feces is largely avoided.

All other measures taken for the protection of the newborn are directed chiefly toward the prevention of the growth of infectious organisms upon the skin and mucous membranes. The success of these efforts depends upon two factors: first, absolute cleanliness in all procedures carried out upon the child; and, second, the maintenance of a normal, healthy condition of the skin and mucous membranes, particular care being taken to prevent traumatisms, however small.

FIG. 11.



Flick's apron-bandage.

As to the first, the bath is of paramount importance and should certainly be given daily from the time of birth, both for cleanliness and for the hygiene of the skin. The temperature of the bath should be prescribed by the physician at 35° C. (95° F.), to be ascertained by a bath thermometer. The temperature should be neither higher nor lower than this. After the first six months the temperature may be ordered at 33°–34° C. (91°–93° F.). Careful drying of the skin after the bath is of great benefit in keeping it in good condition. In the middle or at the end of the second year the temperature of the bath may be gradually lowered to 31°–32° C. (88°–90° F.), and after that may be slowly reduced a few degrees from year to year. Baths in flowing water should not be permitted until the seventh year. In addition to the bath, daily local washing with mild soap is desirable, particularly of those parts which are liable to become soiled or where free perspiration occurs (as between the fleshy folds of fat children, cleansing them if possible before irritation takes place). This is particularly necessary in the region of the neck, where milk is apt to trickle during nursing, and in the

axilla, the bend of the elbow, knee, and groin, and the anogenital region. The child should also be cleaned after each change of napkin, which latter cannot be done too often or too carefully. The head should receive particular attention, as not infrequently an excessive dryness of the scalp causes it to become covered with a layer of epithelial debris and dirt particles, which may lead to a chronic eczema of the part, but which is easily prevented by the timely use of soap followed by an application of oil.

The eyes of the newborn infant also demand certain preventive measures. As infection of the eyes may occur during the passage of the child through the genital canal, it is well to make this region as surgically clean as possible by means of vaginal douches before labor. After birth the eyes should be washed with clean absorbent cotton and water set aside for that purpose, followed by that best of prophylactic measures, a few drops of 2 per cent. silver nitrate solution instilled into the conjunctival sac, as advised by Credé. In case one eye becomes diseased, the other should be protected by a cover or bandage, and the strictest aseptic measures should be observed, in order that other members of the family may not become infected through the use of wash-bowls, towels, etc. As in the case of the eyes, so may the nose, mouth, digestive tract, and genital apparatus become in a similar manner the point of entrance for the invasion of micro-organisms, making it necessary to cure, as soon as possible, a local lesion wherever existing.

Thus it is readily seen that in nearly all the orifices of the body, careful cleansing is the best prophylaxis. There is but one exception to this general rule, and that is the mouth of the newborn and of the infant, for the reasons given in the previous pages. As it is well-nigh impossible to wipe out the baby's mouth without producing slight superficial lesions, the washing out of the baby's mouth before and often after each feeding, formerly a common practice, has been entirely abandoned. Upon these lesions the bacteria may settle most readily, whether normally present in the mouth, or introduced upon the cotton or linen used for wiping. The presence of Bednar's aphthæ may occasionally be the starting point of a general infection, or the cause of serious constitutional trouble. These facts present such unanswerable arguments against washing the mouth, that the benefit to be derived from the mechanical removal of the milk coagula and the schizomycetes to which they lead, need not be considered.

While the maintenance of a healthy skin and mucous membrane is of prime importance in the first days of life, in order to prevent the entrance and possible spread through the blood current of pathogenic organisms, which may endanger life; it is still more important in the succeeding period of the infant's career to prevent digestive disturbances by rational methods of feeding, so that no retardation or cessation of

development may occur, nor any of the conditions may ensue which if not checked frequently prove fatal. The general care of the infant in this respect is naturally most important, cleanliness and neatness taking a leading part therein. This care comprises not only the bath, the handling of the linen, the furnishing and arrangement of the baby's bed, the nursery and attendants, but also the preparation of the food and all the procedures which I have already briefly sketched regarding the prophylaxis of the newborn.

The results of this care of the infant will stand or fall according to the intelligence and training of the nurse. The training of good infants' nurses who know exactly what is required for the proper care and correct feeding of the baby raises a protecting wall against the foolish advice of ignorant midwives; and the direct teaching of those mothers who cannot or do not have nurses is the most effectual means of preventing the diseases of infancy. The nurse, as well as the physician, must understand the care and management of the infant. She must know how easily most cases of intestinal catarrh, simple cough, and coryza can be conveyed from one infant to another by contact; that persons and utensils are sometimes instrumental in carrying the infection; and how dangerous to the infant many of these mild conditions may become. In modern infants' hospitals isolating walls (Grancher's boxes) are used between the beds of the patients, in order to prevent the spread of infectious germs which may exist upon inmates or surroundings. In regard to further precautionary measures and the carrying out of asepsis to diminish the danger of infection by contact in hospitals, I refer you to Heubner's fundamental work upon "Infant Feeding and Infants' Hospitals."

In the family, also, prompt isolation of the sick should be practiced for the protection of the well. Beside infection through contact, sensitive infants may become infected through the air. Inasmuch as the finest dust may contain bacilli-laden particles, carried from the sick, this forms a very common source of infection for healthy individuals. In this manner a child may infect another, or the nurse, mother, brothers, and sisters, when suffering from an affection of the upper respiratory tract (*e.g.*, rhinitis, pharyngitis, bronchitis), are likely to carry the disease to the infant. Whenever, for any reason, it is impossible to remove from the vicinity of the child a parent or nurse suffering from catarrhal infection, it is well for her to wear the well-known Mikulicz's face mask.

Aside from the foregoing precautions, the healthy development of the infant at this period depends upon rational nourishment.

It remains, at this point, to advance all the rules and measures presenting the greatest advantages in the rational feeding of infants, this being the best prophylactic against disturbances of the digestive apparatus, and consequently an agent in reducing infant mortality. These

rules and measures may be found in detail in the chapters on "Milk" (Raudnitz) and "The Nourishment of the Healthy Infant" (Camerer). I should like to proclaim but one thing, which is of supreme importance in the study of the feeding of infants and the prevention of gastrointestinal diseases at this age, and that is *that there is only one rational food for the baby, and that is the natural mother's milk*. All our efforts and striving have given us but a very inferior substitute. When the truth of this proposition is recognized, the physician who is interested in the hygiene of children and the prophylaxis of their diseases will do his best to promote this phase of infant feeding wherever he can. He must also work to the end that the young and rising generation of women may be properly trained to their future destiny of motherhood through plenty of healthful exercise and through the abandonment of tight clothing which may press upon the breasts and interfere with the functions of the mammary glands. By instructing the mother, in the later months of pregnancy, as to the proper treatment of the breasts, the physician may accomplish much in the way of developing them and obtaining richer milk. It should also be his duty, whenever the opportunity presents itself either in his practice or in other circles, to urge the desirability of this method of feeding the infant, so that those interested may be made to realize that mother's milk is the only proper food for the baby, and one which it is almost always possible to provide. The young mother should be told that it is her sacred duty to nurse the child, and the physician should exert all his energies in assisting her to this end; and he should protest with equal energy against the not uncommon inclination to avoid this duty for social or supposed æsthetic reasons.

The physician should make his field for spreading this teaching as wide as possible by preaching the great benefit of natural nourishment, and by making sure that midwives and nurses acquire more experience and a clearer understanding of the importance of this question; and he should see that the authorities energetically insist upon the enforcement of their duty in this regard, by bringing them under greater and stricter control. He should also exert his influence with the authorities, urging upon them the necessity of proper pecuniary provision for poor women who are willing and able to nurse their own infants, but whose poverty forces them to go out as wet-nurses, and thus to sacrifice the health and life of their own infants in caring for the children of wealth. Here is an opportunity for philanthropic societies, to provide these "wet-nurses" with sufficient means of support for at least several months, not in the way of charity, but as proper compensation for the maternal duties which they assume. As a good beginning for the spread of this propaganda, the physician should establish stations for the care of infants, thus arousing the interest of the municipality, the state, and private societies, in the questions of infant feeding and the

causes of high infant mortality. As time goes on and his teachings spread to a larger and larger public, his task will become lightened, and wider attention will be directed to the mother's prime duty.

But as the possibility for breast-feeding is not present in every case, the artificial feeding of infants must always remain as a necessary evil. In such case the watchfulness of the physician must be many-sided, for the fact is that mortality and morbidity are far greater among artificially-fed children than among those nourished at the breast. In order to prevent the ills induced by artificial feeding, the physician himself must wield all the experiences gained and advances made during the last twenty years in the study of the organs of nutrition and of the metabolism of the infant. Simply stated, success depends above all upon our ability to give an unimpeachable (aseptic) milk. The step next in importance is to preserve the aseptic character of the milk from the moment of milking until it reaches the child. This is accomplished by keeping it cold, by handling it in the proper manner, and by protecting it from contamination. In this matter also well trained midwives and nurses can prevent much trouble, and the physician can be of great assistance through his advice given at the stations for the care of infants (Fürsorgestellen). The physician must, of course, possess the experience necessary to enable him to cope with the difficult problem of properly arranging the food formula for each individual case. This done, everything depends upon the proper quantity and frequency of the feeding.

If the physician, both as teacher and as adviser, will concentrate his efforts on the prevention of gastro-intestinal diseases, with the consequent reduction of infant mortality, he may be certain that he will have accomplished a great work.

In addition to the dangers by which, through disturbances of digestion, the child is threatened in infancy, it is in danger of one particular disease which, although it may occur at any period of life, seems to have a special predilection for the very earliest days of childhood. This is tuberculosis, and particularly tuberculosis of the bronchial glands. Only a small proportion* of infants become infected through germ-laden nourishment, and a still smaller proportion by swallowing the germ of the so-called "feeding tuberculosis" (Fütterungstuberkulose). In order to prevent this disease, it is necessary that the infant obtain a milk free from tubercle bacilli. Under no circumstances should it be offered the milk of a mother or a wet-nurse suffering from tuberculosis. The greater number become infected through the inhalation of tubercle-laden dust, or by direct contact during the acts of coughing, speaking, kissing, etc.

The infant should be kept from the vicinity of those suffering

*There are very few cases of infants born with tuberculosis.

from tuberculosis, even though they be the parents or members of the family, and it should not be permitted to grow up in their society. The separation of an apparently healthy infant from the tuberculous family and its transplantation into a healthy family, although it is an important prophylactic measure, is possible only under extraordinary circumstances. In spite of this, however, the physician should always attempt to separate the healthy infant from the diseased. Well-to-do parents, if they desire it, can readily find a place for their child in a healthy family or in an institution in the country or elsewhere, by paying for it. But the poor can also find it possible to remove the child from its unhealthy home influences through the care of modern sanatoria and recuperating places. Should it be impossible to carry out this project, however, it then becomes necessary to call to our aid all the prophylactic measures which modern hygiene has created, to prevent the spread of tuberculosis and to overcome it. Special care must be taken to prevent the scattering of tuberculous germs through spray from the mouth or from sputum; these germs, after becoming dry, are mixed with the dust and are then taken up by the air currents. The walls, and particularly the handkerchiefs, must be disinfected. Those suffering from tuberculosis should not be permitted to kiss others, to use the same dishes, or to taste the food intended for the infant or to blow upon it in order to cool it off.

We will now leave the infant and take up early childhood, the period before children go to school. During this period the health of the child is threatened principally by two sorts of dangers. The one passes for a picture of a more or less well marked indigestion ("spoiled stomach"), which is brought about by improper, irregular, or too rich meals. The other danger comes through the usual infectious diseases which show a preference for this age, and which, owing to their frequency, really make up the so-called children's diseases. In the second and third years the physician will not particularly meet with the fevers, and he will be consulted with regard to feeding much more rarely than during the first year. And yet as family adviser, he can prevent much trouble during this period, by insisting that regularity be still maintained in regard to meals, and that the child must not receive things difficult of digestion, particularly sweets and dainties, which when dealt out in small quantities as a reward after the meal are not harmful, but which when given in larger quantities act as an irritant, and should be avoided. At this age, through irregularity in the nourishment, there frequently develops that common and stubborn trouble, chronic habitual constipation. This can in many instances be certainly overcome by dietary rules and instruction in regard to suitable physical hygiene and exercises.

It will be less easy for the physician to guard the health of the child

against infectious diseases. This inability is inherent in the nature of the case. Nearly all our infectious diseases, not only in the large cities but also in the country, have become endemic and therefore can no longer be uprooted. The possibilities of transmission rest not only with the patient himself but also with third persons, and they are so many that it is very difficult to guard the child against the disease. The danger of contagion is present every time the child goes walking, goes in any conveyance, or plays with other children. Owing to the severity of many of the infectious diseases and the seriousness of the situations to which they may give rise, and considering the great weakness of the young organism, it is naturally of importance that we postpone the infections as long as possible, even if we cannot prevent them. Occasionally one hears or reads of families in which the children were brought up isolated from all other children, and were thereby enabled to reach the age of puberty or beyond without ever having had any of the severe infectious diseases. In most cases, however, stringent precautions are taken only during an outbreak of any one of the infectious diseases. Still we should insist upon the enforcement of the order to avoid persons suffering from infectious diseases, as well as healthy persons who live with or have visited a sufferer from such a disease, so that the child may be spared this danger, the consequences of which, so far as its life is concerned, can never be foreseen.

Where a contagious case exists in a household, prophylactic measures offer a better prospect of success in preventing the spread of infection, than in attempting to actually guard against it. The physician may obtain splendid results in such a case by the strict enforcement of hygienic measures of various kinds. Briefly summarized, these rules depend upon the rigid isolation of the sick from the well, which is best accomplished by the removal of the patient to a hospital or sanatorium; the complete shutting off of the sick room, the nurse, and the kitchen used by the patient; disinfection and the careful handling of the excreta of the patient; and, after the disease has run its course, disinfection of all the effects, toys, linen, dishes,—of everything, in fact, which may have come in contact with the patient.

Particular care must be taken in scarlet fever, diphtheria, and whooping-cough. Scarlet fever is contagious through the desquamation of the hands, for weeks after the actual symptoms have disappeared, and the parents should be warned of the possibility of conveying the disease by mere handshaking, or in the course of play. As it is impossible to prevent children from playing with their fellows after a prolonged period of isolation, and as they may be contagious in no other way except through the hands, it is obvious that the wearing of easily washed and easily disinfected white cotton gloves would prevent the transmission of the disease.

We know that in diphtheria, long after the local process has disappeared, virulent diphtheria bacilli may be found in the mouth. The possibility of contagion exists for a longer period than that during which we are in the habit of isolating the cases. An effort must therefore be made, even after the usual isolation period of six weeks, to prevent infection by the mouth through contact. This may be accomplished in great measure by forbidding kissing.

The same is true also of whooping-cough, which may be contagious for a long time, being transmitted through the mucus from the nose and throat. As is usually the case with infectious diseases in children, this affection is ordinarily transmitted during play. An endeavor should therefore be made to keep these children from coming in contact with healthy ones as long as possible. It is just such an infectious disease as whooping-cough which can most readily be prevented from spreading by carrying out the principles of extreme cleanliness. Mucus from the nose and throat of children readily becomes attached to the fingers, or is conveyed to them from the handkerchief, or is wiped off upon the clothes. It is therefore to be recommended that the children wear easily changed, washable garments, or that the clothes be protected by aprons. Aseptic handkerchiefs are worth while, as they may be burnt up immediately after having been used. The hands should be washed immediately after every paroxysm of coughing. A child suffering from whooping-cough should avoid those places where children are in the habit of congregating, such as open play grounds, or bathing places, which are likely to be sought by children who are recuperating. Not infrequently a popular summer resort or bathing place is selected to which to send a child suffering from whooping-cough, under the erroneous impression that the disease will be cured through a change of air.

Infectious diseases do not belong only to the period before the child goes to school, since the period of school years claims further victims from among those children who have been previously shielded from contagion. As the school itself is very often the medium for spreading disease, the physician must, at the beginning of an illness accompanied by any fever, absolutely exclude all infectious diseases before allowing the child to return to school. He should endeavor to impress upon the parents that they should never take it upon themselves to pass judgment upon a case, and that even where the manifestations are mild, it is important to consult a physician. It is common enough to find that influenza, varicella, and the prodromal stage of measles are unrecognized, the children are permitted to attend school, and the infection is spread among the pupils. The general practitioner can do much to further the cause of the prophylaxis of infectious diseases by instructing the families, to their great benefit.

There rests with the physician an important duty in the way of prophylaxis, in the prevention of the sequelæ of the infectious diseases. Even though he stands helpless in the face of many complications which occur at the beginning or during the course of the disease, he is nevertheless justified in anticipating certain troubles (as severe anæmia, or tuberculosis) after such diseases as whooping-cough and measles. These can often be avoided through good nourishment, careful observation, and the application of all the hygienic measures at our disposal, particularly fresh air and change of climate. Even for the poorest, in Germany there is ample provision in this regard to be found in the recuperating and cure places and the woods schools.

With the entrance into school, a new life begins for the child. The open air life must of necessity give way to the requirements of school-time. The harmless, untrammelled play must, in a similar manner, be repressed; and opposed to the unrestricted hours are the method, discipline, and restraint of the school. It is "sit still and pay attention!" Here arise new conditions for the young child, conditions which are entirely different from the earlier ones, and which cannot possibly be without influence upon its body and mind. These changes in the entire scheme of the child's life, and the possible evils resulting therefrom, must not be blamed altogether upon the school, as the disturbance of all the usual relations is necessary and natural. The agitation aroused in the young organism by the requirements and new impressions of school life can best be allayed by instituting a healthy routine.

During this period the physician must be especially careful to ward off the injuries which school influences may produce upon the health of the child. Among the poorer classes, the school physician now takes the place of the family physician in the supervision of the child's health; but, although the duty of watching over the physical and mental welfare of the child during school years is obligatory upon the school physician, it should none the less remain, at least in part, the duty also of the family physician. Both must be careful that children who are physically or mentally backward, whether from chronic diseases (rachitis, anæmia, scrofula) or otherwise, should be closely observed upon entering school, to determine whether or not they are able to keep pace with normal children of their own age. Should it be decided that they are not up to this standard, they should at once be placed in a lower grade, not only in justice to themselves, but also in the interest of their class mates. This period should then be devoted to so caring for the health of the child that it may finally reach normal development. Serious defects or illnesses (deafness, blindness, imbecility, epilepsy, tuberculosis) require special schools or institutions. It is exceedingly important, also, that the sight and hearing of the child be carefully tested before its admission to school, so that any abnormalities may be neutralized or corrected

by giving the child a most advantageous position (toward the front of the room) in the class room.

After school life has begun, it is essential to see that the injurious effects of the new conditions (sitting still, impure air, close attention, fear, mental overstrain) are reduced to a minimum. The harmful effect of daily attendance at school shows sooner or later; but it is principally apparent among the very young, or delicate, nervous, or anæmic children. The general term applied to this condition is "school sickness." Its manifestations are varied and numerous, being sometimes one, sometimes another, of the following symptoms, or sometimes a combination of several: headache, lassitude, anorexia, insomnia, irritability, nervousness, severe anæmia, and hysteria. This condition is, without doubt, a sequel to the alteration in the child's mode of living. Even if the physician is unable to prevent this condition in all cases, he is at least in a position to see that no serious injury results therefrom. If the hygienic arrangements of the school are satisfactory, much will have been accomplished. Large, airy rooms, with the best facilities for ventilation, should be insisted upon, not only in the public school, but in private schools as well.

The floors of the school room should be smooth, and oiled to prevent the collection of dust. The temperature should be between $17\frac{1}{2}^{\circ}$ and 20° C. (64° – 68° F.) and should be regulated according to the indications of a thermometer. The best method of heating is by means of the hot-water system. The benches should not be too near the radiator and should be so placed that the light, from windows as broad and high as possible, falls upon them from the left. Artificial light should be used as little as possible. Proper school benches are of the utmost importance. In addition to these requirements in regard to the building, the physician should be consulted with reference to the amount of school work, the number of school hours, the recesses, the distribution of vacation periods, etc. If all these conditions, and such others as may arise, are hygienically attended to in turn, most causes of the harmful effects of school life will have been removed. The general practitioner, as well as the school physician, may accomplish much in this respect. Let us hope that in future the physician will be consulted upon the question of removing from the school certain sick children (especially those suffering from tuberculosis) whose presence is a menace to the other scholars, thus attaining a distinct advance in the prophylaxis of diseases occurring during school years. In like manner, the physicians should be permitted to formulate new rules as to when a child may return to school after recovering from an infectious disease, or when a member of its family is suffering from such a disease.

By instructing the teachers on the subject of hygiene, much can be accomplished in the way of correcting defects of vision, injurious atti-

tudes, and other ills resulting from or aggravated by school work. Such instruction is now being given in many places for both teachers and parents, and these meetings result in the exchange of much useful advice and many helpful suggestions tending toward the improvement of these conditions. In carrying out these measures and precautions to preserve the health of the child during school life, the physician may rely upon the devotion and interest of the teacher. He must, however, so regulate the time spent by the child outside of school that there may be a minimum of interference with the course of instruction.

To attain this end, the physician should call to his aid all possible hygienic measures. In order that the child may properly perform all its new duties, it is of the utmost importance that its health be kept as nearly perfect as possible, and that its physical strength be developed by exercise adapted to its age and sex, such as swimming, skating, exercise in the gymnasium, play (basket-ball, foot-ball, lawn tennis, etc.) in the open air, and, last but not least, plenty of walking. In order to prevent the tiring of either mind or body, the day's activities should be suitably distributed. The hours of rising and retiring must be carefully regulated, and ten hours of rest at night should be insisted upon during the earlier school years. It is advisable, for many reasons, that absolute regularity be maintained in the hours for sleep, permitting no deviation even on Sundays and holidays. With such deviation there develops a tendency to lie awake in the warm soft bed, which frequently arouses erotic sensations and which may finally lead to onanism. It is preferably to be recommended that the spare time of the child be devoted principally to long walks or to some congenial occupation for which there is no time during the week. The day itself, too, should be so divided that there will be regular interchange of work and freedom. In order to insure complete mental relaxation, the periods of freedom should be sufficiently long to permit the taking of walks, or the playing of games.

The nutrition of the child during school years should particularly engage the attention of the physician. It is striking that, just in the early school years, there is a flagging of the nutrition. This may be due, in part, to the fact that the children, being unaccustomed to early rising, laboring under the excitement of an effort to reach school punctually, and fretting for fear they may be late, cannot eat breakfast comfortably in the time at their disposal. It is not uncommon to find a failure of appetite after a time among those young children who go to school without having eaten enough. They do not care for the luncheon they take with them to school, and are likely to partake sparingly of the mid-day meal or to refuse it altogether. In such cases the physician can enforce prophylactic measures by instructing the parents that it is necessary to insist that the child eat a plentiful breakfast; and by showing them that the smaller the quantity of food and the less frequently

it is introduced into the stomach the less the desire for food becomes, until finally there is complete anorexia; but that after a good breakfast, the child is apt to hunger for luncheon, so that finally the appetite will

FIG. 12a.



The Charlottenburg school in the woods—the mid-day meal.

become normal. The quality of food appropriate for this period should be considered, as well as the regularity of the meals. This should consist principally of fats and carbohydrates, and, in preference to the

FIG. 12b.

The Charlottenburg school in the woods—the singing lesson
in the open air.

meat and eggs which the parent is likely to offer under the impression that they contain more strength, the child should receive plenty of fruit and vegetables.

Those children who possess certain constitutional anomalies, or who are delicate or anæmic, demand special supervision from the physi-

cian, so that they may not be harmed by overtaxing their strength at school. Where the strain is telling upon body and mind, the child should be relieved of those studies which are not absolutely necessary for

FIG. 12c.



The Charlottenburg school in the woods—open air play.

its advancement, such as drawing, manual training, singing, technical branches, etc. This can be accomplished through a physician's certificate as to the necessity for this action. Extreme watchfulness should be given

FIG. 12d.



The Charlottenburg school in the woods—the close of school.

to the work in the gymnasium, and special work in this line should be given to children who have hernia, or a tendency to hernia; and to those suffering from tuberculosis, bone disease, lameness, or fractures, as well as to cases of scoliosis, for which orthopædic gymnastics are required.

But in certain individual cases even relief from part of the school work does not suffice, and it then becomes necessary to discontinue school work entirely, or to give instruction but a few hours daily, either individually or with children of similar limitations. In this direction a brilliant vista opens up for the benefit of sickly children, as is illustrated at the "School in the woods," in Charlottenburg, which has already been taken as a pattern by other cities.

FIG. 13a.



Bad position while writing
(after Hoffa).

FIG. 13b.



Bad position while writing
(after Hoffa).

The posture of the child and the condition of its vision must be particularly considered in the prophylactic measures instituted by the family physician. The attention of the parents should be directed to faulty positions assumed by the child, such as sitting with the body twisted or "crooked," the shoving forward of the right or the left shoulder while writing, or the advancing or dropping of one hip while standing, so that by constant correction the resulting evils may be prevented. The physician should personally correct the faulty attitude of the child, lest through lack of early attention the time should pass when a possible scoliosis may be prevented; and in order that the muscles of

the back may not remain inactive, thereby becoming the first step to a faulty posture, as is so frequently the case.

The child should not be kept too long in any one position (sitting, or bending over as in writing), but should be frequently interrupted, say at half-hour intervals, and be made to sit erect, or to take short marches, or calisthenics, or some gymnastic exercises.

Memorizing is most efficiently accomplished while standing, walking, or going up or down hill.

One of the most important factors in the prevention of scoliosis is a proper writing desk for the use of the child. The ordinary desks

FIG. 14.

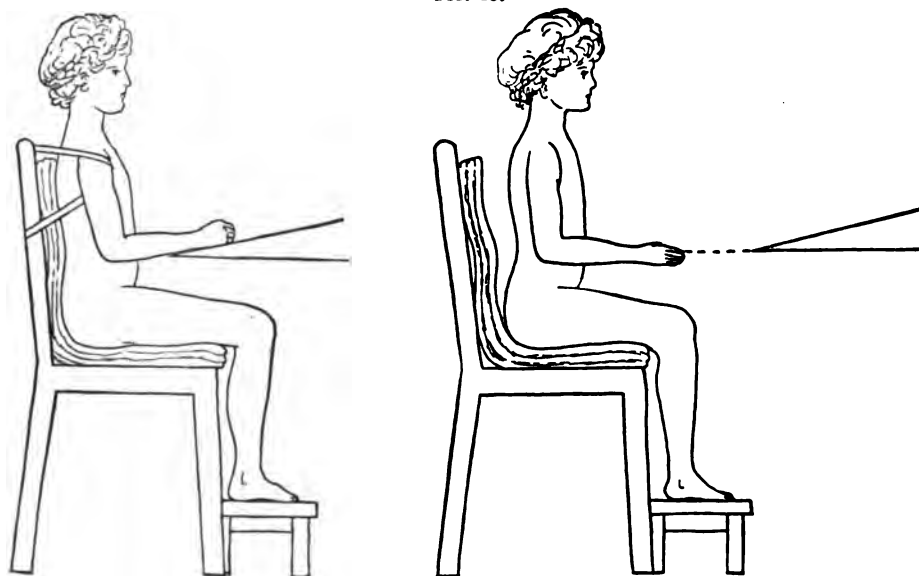


Work desk. Normal position in sitting.

found in the market, in spite of numerous improvements, leave much to be desired, for if the desk is not suited to the size of the child, it will do more harm than good. An ordinary table and chair are often preferable, each bearing the proper relative height to the other, and to the size of the child; and both being so constructed as to afford the child a comfortable upright position while seated, without producing cramp or fatigue. The height of the bench or chair should equal the length of the child's leg, the entire sole of the foot resting upon the floor. The depth of the seat should equal three-quarters the length of the thigh. The height of the table should be regulated according to the height of the

chair, the vertical distance between the edge of the table and the anterior edge of the chair being, in boys, one-sixth to one-eighth the length of the body, and in girls $1\frac{1}{2}$ to 3 cm. more. The top of the table should be level with the child's elbow when seated. The edge of the desk in the rear should be about 2 to 5 cm. higher than the top edge of the bench in front to permit the correct position in writing (Minusdistanz). A skilful carpenter can make an ordinary wooden chair and table fulfil all these requirements for the correct seating of the child, by adjusting them in the proper relation to each other and to the child. The "Minusdistanz" is readily ascertained by shoving the chair under the table for a short distance. Another method of preventing scoliosis is the intro-

FIG. 15.



Normal position while writing, seated upon an ordinary chair.
(after Lange, Munich).

duction of vertical writing (Steilschrift), practiced in the most radical manner, with the lower edge of the paper parallel with the edge of the desk.

The physician must instruct the parents regarding the harm which may result to the eyes by using them in the twilight or in artificial or poor light, or by reading small writing, poor printing, etc.

To prevent the morbid conditions, especially of the nervous system, which manifest themselves in muscular relaxation and anæmia, it is necessary to exercise all the measures mentioned above: a simple but nourishing diet; rational development and cultivation of bodily strength; sufficient periods of rest after working; fully satisfying the requirements for sleep; plenty of time spent in the open air; and filling up the hours of freedom with childish play and sport. Special emphasis should be

given to the importance of a healthy bringing up, which should include as its most potent factor a good example, and in which due regard has been given to the child's talents and individuality. Just as the prophylaxis of the child's physical life demands the sparing of its powers

FIG. 16a.



Position of children when writing vertical script
(after Hoffa, Berlin).

and their use in moderation, so are the same measures necessary to prevent overtaxing of its mental strength, and for the conservation of its spirit.

In regard to these latter, it is possible to make only a few brief suggestions. During the first year of life, it is proper and necessary to

FIG. 16b.



Position of children when writing obliquely
(after Hoffa, Berlin).

give the child absolute mental and physical rest, and not to interfere with its tendency to sleep, because sleep and rest mean development for it. Nor should we attempt in the second and third years to arouse its mental powers by questions and stories, in an endeavor to produce precocious development. During this time, it is merely a matter of

fostering the senses; and, inasmuch as the spiritual life is built upon sensory impressions, everything depends upon the proper development of the senses and the reception of correct impressions obtained in the proper strength.

If, during the third and fourth years of the child's life, it asks continual questions, it is absolutely essential that all these questions be correctly answered, with no further detail or explanation than necessary and with due consideration to its limited powers of comprehension. If the question is answered correctly and concisely no false and unreal conceptions will be developed in its mind, and the child's imagination will not move upon false lines.

After a time, of course, there will come to the child a thirst for knowledge, followed sooner or later by the emergence of the mighty instinct for occupation, for imitation, and for the society of its kind. To direct and guide these so that they shall move in proper channels and shall not be followed too intensely, requires the most solicitous thought and observation of the parents. In this way, by careful individualization, the early training will make for a strong and healthy sentiment, and for the establishment of strong will power, of self control, and of the ability to bear pain and disappointment.

Particular adroitness must be exercised in answering questions regarding the sexual life. The physician should assist the parents by his advice upon this subject, or, if opportunity offers, should give the child at the right time a natural explanation of the question, which will bring it into harmony with the spiritual life of the maturing child, and make it of inestimable value during its years of development. In which manner, at what time, and how best to approach the sexual question, I will not discuss here. If the parents through proper bringing up have gained the confidence of the growing child, it will generally, and particularly in this matter, sometimes earlier and sometimes later, come to its parents, usually the mother, and freely discuss the question. Its queries should be answered openly, honestly, and naturally, with proper regard to its mental development and its ability to comprehend. If this is done, the harm which results from confused ideas and lustful thoughts may be prevented, and the practice of onanism and masturbation reduced to a minimum. There are many other measures which may be taken in the care of the mental and spiritual development of the child, which I will touch upon only superficially: these are, guarding against satiety (avoidance of gifts and toys of too costly a character and of too great a number); care as to arousing too early the mind and imagination by premature and too frequent participation in children's parties, musical performances, and the theatre; the avoidance of too continuous an association with adults; and, the prevention of the reading of evil and unhealthy literature.

Another subject to be considered by the physician is the occupation of the child during its spare time, or of any special branch of knowledge to which this time is devoted, either through talent and inclination, or by the wish of the parents. In this category are included music, drawing, painting, and handicraft. In order that the later life of the child may not be hindered through injury to the mental state by overburdening and overtaxing its mind in these early years, due consideration should be given to its age and the proper hours for the pursuit of these and equally important occupations. The physician should also determine whether, in particular instances, the child is not so overtaxed by school work and the consequent home studies as to make it necessary to give up some of them, or perhaps to abandon them for a while and dispense with the school altogether. This relief from school work should also be insisted upon when necessitated by excessive mental strain, or when the tax upon the physical strength is too great, as I have pointed out elsewhere.

For the psychical equilibrium of the child, upon which the foundation and steadfastness of its character are reared, there is nothing of such extreme importance as the good example of the parents. Self respect and good precept are the mainsprings of a healthy and effective method in the bringing up of children. Example is more potent than are exhortation and correction; unity and peace in the household produce equanimity and harmony in the child; indecision and discord, on the other hand, produce uncertainty and heedlessness. Quarreling and scolding frequently call forth, in sensitive children, a feeling of sadness and loneliness, which may even lead to a state of melancholy. The family physician who comes and goes in such a household has truly not only the physical but the spiritual needs of the child to look after, so that there may be a healthy foundation upon which the intellectual and moral being may develop. The totality of the spiritual training is to be found in a complete, harmonious development of all the psychical faculties, the powers of observation, of discrimination, of memory, of will, of sentiment and soul, and an appreciation of the noble and beautiful.

The foregoing chapter upon the general prophylaxis of childhood can, naturally, merely touch upon many of the injuries and dangers which may threaten the child's health during the various periods of its life. It has been possible only to hint and suggest regarding the care to be taken. More specific rules and details will be found in the special prophylaxis of the individual diseases, and in the chapter relating to their treatment.

GENERAL THERAPEUTICS IN DISEASES OF CHILDREN

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FOR a long time to come, it will be easier to prevent diseases than to cure them.

It is true that during the last decade, with the increase of our clinical knowledge, which has been accompanied by a refinement of diagnostic methods and a better understanding of the etiology of disease, some progress has also been made in the treatment of disease. But in spite of all this, the great and intricate apparatus of our modern therapy must even now be virtually content to improve and alleviate the condition. When, through disease of a greater or lesser organ complex, the physiological limits have been transgressed, it becomes the task of therapy in general to cause a return to those limits. The stimuli which are supposed to be the cause of all vital energy are capable of perceptible increase or diminution. It would be necessary merely to discriminate between the unnatural and the natural stimuli, were it not for the evil influences of the environment with which, for all time, we have been surrounded and which therefore, up to a certain point, become physiological. These influences are not to be looked upon solely as a menace to the human organism; but also as an agent for hardening and elevating the powers, especially during the period of development.

In the practical application of the foregoing, by means of the etiological factors, we will always differentiate between extraneous chemical and physical stimuli, and an abnormal increase or diminution of physiological stimuli. Thus, the functions of the intestines may be disturbed on the one hand by the ingestion of decomposed food or even poison, and on the other by an overloading with ordinary food. In the one case the foreign ingesta will be removed or neutralized, and in the other the usual supply of food will be decreased. The principle involved in the removal of external irritation, of either a chemical or a physical nature, being readily understood, it is unnecessary to dwell upon it further.

The treatment of functional abnormalities (to which, after all, it is possible to refer all diseases) must take into consideration the following limitations of therapeutic stimuli:

In the first place, the functional activity of the developmental period of the human organism must be regarded differently from the later period. The sensitiveness of the intestinal tract in infancy, and of the nervous system in infancy and at the time of puberty, should be remembered.

Secondly, owing to heredity, it is not infrequently the case that the functions of certain organs or systems are from the beginning irritable in an abnormal degree, and these may show morphological changes, or even degenerations. The nervous system furnishes particularly good examples of functional incompetency.

Thirdly, the irritability of a system may be entirely changed from the normal, whether through habit or as a result of disease, without the system necessarily being functionally incompetent. This is illustrated by the skin, where a tendency to sweating may occur, owing to exposure to the heat of summer, or to the various procedures used in the production of diaphoresis, or after rheumatism. This tendency is apt to create a sensitiveness to a rapid cooling off; but, on the other hand, by instituting proper measures for hardening the system, this can be overcome.

Fourthly, and lastly, the abnormal irritability which exists during disease must be taken into consideration, in so far as it may influence the strength of the reactions of the therapeutic stimuli. Disturbance of function manifests itself, as a rule, by an increased irritability, whereas a paralysis of function is the forerunner of certain death of the organ. In the stage of diminished irritability, stimuli which ordinarily have no perceptible effect that can be roughly demonstrated, may under certain favorable circumstances have a distinct action, usually physiological; and, in a more marked degree, abnormally strong stimuli may injure or even destroy function.

The same form of irritation may quiet, excite, or paralyze the action of an organ, according to the size of the dose. This point must be given equal attention, whether the treatment be dietetic, physical, or medicinal. A morbid increase in function does not as a rule lead to an increase of the combined activities of the organism. Treatment will usually diminish the function in the beginning. In general, then, the lessened activity of the organ will result in its regulating its own activity. Later, this is accomplished by diverting work to other systems, whose activities become greater. The removal of the irregular, disease-producing stimuli, on the other hand, permits of an increase of the functional ability of the diseased organs through use. It is particularly characteristic of the youthful organism that its functional ability fluctuates in far greater amplitude around the normal than does that of the adult, inasmuch as its activities may be considerably increased without injury. The organism, by making use of this, brings about a permanent augmentation of its capabilities. There is also a far greater power of regeneration in the diseased organs than exists in the adult.

Even though the therapist is compelled to hold to these theoretical explanations, they do not give him any information regarding the measures which should be taken. The mutual relations of the different functions of a single organ, and the relations which exist between the functions of the different organs, are too much involved to allow their scientific basis to be of much use for practical deductions. Since therapy as a rule emanates from empiricism, confusion and uncertainty will exist for a time; meanwhile, the estimation of individual methods of treatment fluctuates between high favor and neglect.

To him whose highest aim continues to be the curing of the sick, however, it would be as difficult to give up the striving for scientific knowledge as it would be for a lover of theory to give up a remedy which has given him good results, because its mode of action is not thoroughly understood. Careful observation must, in every instance, ascertain the mode of action and the strength of the remedy, due consideration being given on the one hand to the strength of the drug and method of administration, and on the other to the age and individuality of the patient and the character of the disease. As a rule, drugs should not be combined in such a manner as to interfere with the action of any one of them. The desired insight into the action of the treatment is usually more readily obtained with physical than with chemical methods; and in any case, with the latter one should avoid unnecessary and uncertain combinations. There are no remedies which are harmless in all cases. When a remedy is in general capable of causing irritation, it is possible, by using too large a dose in relation to the reactive powers of the patient, to injure the vitality of the organism. This holds true not only for chemical and physical remedies, but for physiological stimuli (atmospheric influences) and for external stimuli generally.

In childhood, much more than in adult life, does the success of a favored method of treatment depend upon the accidental surrounding condition of the patient. For example, it is possible to obtain the effects of a drug prescribed for internal use only if the drug can be administered so that it will be retained and not be expelled by vomiting. Even then failure may occur if the taste of the drug is such as to cause loss of appetite. The successful treatment of sick children is possible only through broad and practical experience in this department, reaching beyond the limits of mere care of the patient. A more intimate knowledge of the child's mental state, and its practical application in the field of pedagogy is particularly important. In the treatment of the child, the physician is dependant upon the coöperation of the adult who accompanies the patient. If ignorance is the greatest danger to health, then it is present here in a most marked degree. In just the measure in which those who surround the child can be freed from bigotry and error and can be led to the observation of the objective symptoms, the treatment

will be sought at the proper time, and the measures will be carried out according to prescription and continued for a sufficiently long time. The physician can and should, during the treatment of the child, do his share by advice and practical instruction in this regard.

We will now take up the general subject of the treatment of children's diseases, in the following regular order: Nourishment, Nursing, Psychotherapy, Physical Treatment (particularly climatic, and hydrotherapy and balneotherapy), Mechanotherapy, combined Physical Therapy, and, finally, Pharmacotherapy.

Nourishment.—Nourishment is an important factor, not only in diseases of the gastro-intestinal tract, but in a greater or less degree in all diseases. Very young, and feeble children, particularly when ill, suffer from defective or deficient nourishment. The general condition is improved by an abundance of food, which produces an improvement in the circulation and innervation, and an increase in metabolism, and causes the secretory and excretory functions to approach the normal. The nutrition of one organ being improved, the diseased organ often shares in it, and in this way is supported and is helped to fight against the harmful effects of the disease. Moreover, it must not be forgotten that an improvement of the symptoms during the course of a disease or of a disease symptom (such as fever) may not be due to the improved nutrition; but that, on the contrary, the improved nutrition must be looked upon as the indirect result of the improvement of the symptoms. Whenever in such a case there is improvement in the appetite, and the increase in weight begins only after an improvement in the symptoms of the disease, the connection existing between cause and effect is often indistinct, or perhaps cannot be distinguished at all, and the significance of the nourishment may be falsely interpreted.

The appetite fails at once, or, more frequently, after a few days, in the acute fevers, and with this failure the amount of food taken is diminished. With the disappearance of the adipose tissue, and the emaciation of the muscular and osseous systems, if the quantity of water taken is diminished, a certain drying out of the organism occurs; but this is much more the case when, with a decreased intake, there is an increase in the consumption of fluid by the body. While, as a rule, in the acute febrile diseases, a plentiful supply of fluids seems to be indicated to maintain the secretions of the glands and to prevent a stasis of the excretions from the intestines and kidneys, it is not always a good policy to try to prevent loss of weight by increasing the quantity of food, even aside from the fact that a full diet is unpleasant to the patient and but poorly tolerated. A temporary loss of weight within certain limits in a previously well nourished child is hardly a disadvantage, since during convalescence the appetite will be so much greater that the loss will be more than compensated. There is no necessity, therefore, in pneu-

monia, scarlet fever, or measles, for urging and bothering a child to take nourishment; whereas, in typhoid, cerebrospinal meningitis, or suppurative processes generally, where the duration of the disease is uncertain, a good nourishing diet is demanded from the outset. It is hardly necessary to discuss the fact that an afebrile disease unaccompanied by wasting, such, for example, as depends upon some surgical, mental, or nervous condition, requires a moderate rather than a full diet, inasmuch as rest in a warm bed is accompanied by a diminished expenditure of energy. A full diet is not always indicated or of use in the chronic diseases, even though it is frequently advised in a theoretical consideration of the subject. Omitting from consideration that form of idiocy which is characterized by an excess of fat, abundant nutrition cannot bring about an improvement in organic nervous diseases, in neurasthenia, hysteria, or other functional diseases, excepting when the nervous condition is caused or increased by bodily inanition. A highly nutritious diet (suitably planned) forms an essential part of the treatment in a few of the chronic diseases; *i.e.*, infantile atrophy, exhaustion, the nutritional disturbances of older children, and in scrofulo-tuberculosis. It is not to be expected that an immediate curative effect will be obtained in such affections as, for instance, rickets, syphilis, and diseased conditions of the blood. Moreover, the final aim of the treatment has not been attained with overfeeding alone (as shown by an increase in weight). Now and again, however, this is a favorable prognostic sign, since the possibility of increasing the weight usually bespeaks the absence of a serious condition whose outcome will be unfavorable. There are, however, exceptions to this rule.

We will not consider here the question of nourishment in diseases of the gastro-intestinal tract or in the atrophic condition of infancy, but will discuss more minutely the indications for a full diet and the quantity and quality of the food of which it should consist.

Whereas, in diseased states characterized by slow progress and absence of fever the digestive powers may be considered almost normal, excepting when accompanied by gastro-intestinal complications (*e.g.*, tuberculosis); in other conditions, particularly the fevers, the function of the gastro-intestinal tract is probably faulty from lack of glandular action. Practically everything depends upon the functioning power of the gastro-intestinal tract; this manifests itself with greater or less certainty through the condition of the appetite.

The appetite as such, as well as its variations, represents an instinctive guide, which, like so many other instincts, has a deep physiological meaning, although it may fail under certain abnormal conditions, or if improper food is used. Frequently enough, however, it remains correct even when opposed to theory (particularly of a chemico-physiological character), from which we must infer that there is a relationship between

appetite and the needs of the body for food. From the investigations of Pawlow, we know that the appetite, as such, up to a certain degree insures digestion by stimulating the secretion of gastric juice, the gastric digestion in turn starting intestinal digestion. Only very few substances have a similar power without the assistance of the appetite, *e.g.*, milk and broths. We can, then, virtually ignore the appetite only when it is a question of supplying fluids containing sugar, salts, acids, etc., while it is hardly possible to dispense with a supply of nourishment which must first be digested in the stomach. The most natural stimulus to the taking of nourishment is healthy hunger, which, in many diseased conditions, especially those accompanied by fever, is waited for in vain, in spite of an empty stomach, reappearing only with the beginning of convalescence. Loss of appetite due to constipation, a frequent enemy, is the most readily removed.

Where the appetite has failed, there are many ways of stimulating and increasing it, the knowledge of which is of great practical importance. Above all, it is necessary to prepare the food properly and offer it in such variety and at such intervals that it will be taken with relish. Appetizing, stimulating, and nourishing foods should be used, and should be combined with due regard for the individual taste, and for the vagaries of appetite prompted by illness. In this way, it is often possible to give more nourishment than when the diet is arranged upon a purely theoretical basis without taking the patient's tastes into consideration. The well known influence of the mental state upon digestion should be remembered, and an effort be made to awaken cheerful thoughts during the meal, as mental depression from pain, mourning, and particularly from homesickness, hinders it. It is often possible, in the nursery or at the sick bed, to arouse the appetite by a variety of methods; sometimes severe disciplinary measures lead to a quicker and better result than can be obtained by other means. Finally, the use of drugs for improving the appetite is invaluable. As we will explain below, the body weight can be favorably influenced by improving the appetite without changing the diet in any respect.

How the normal child should be fed at the various periods of its life will be considered in another place. The nourishment of the sick child should approximate as closely as possible to that of the healthy one. This is least feasible in the acute febrile diseases, as the food must be greatly diluted and macerated so that the digestive juices may act upon it readily. Vegetables rich in cellulose, meat rich in fats and connective tissue, and fats in solid or liquid form, are to be avoided. Aside from the particular indications for this nourishment in diseases of the intestines (*e.g.*, typhoid fever), it is also of value in prolonged febrile conditions, in which, if the fever is absent for hours or days, these periods free from fever may be utilized for the administration of stronger

nourishment and perhaps of solid food. Moreover, in that most frequent disease of childhood, scrofulo-tuberculosis, if the temperature is carefully taken for periods of weeks and months it will very often be found that the maximum temperature for the day lies at the upper limit of the normal, or it may be subfebrile. A fall in the temperature in such a case will be accompanied by an increase in appetite. It may perhaps be chronic intoxication from the poison of tuberculosis which, in the various forms of scrofulo-tuberculosis and outspoken tuberculosis, lowers the appetite or renders it capricious. Here it is necessary to stimulate the appetite by the use of drugs and by the particular kind of food suitable for the purpose, the consistency and composition of the food not being too unlike that required by a normal child. Of all foods, milk whether raw, pasteurized, or boiled, holds a most important place in febrile diseases, especially as it can be digested in the absence of appetite, owing to its fluid form and its composition, and because it can be obtained in large quantities. In the absence of fever, however, milk should be withdrawn if it interferes too much with the administration of a normal mixed diet (which is to be desired), or if it leads to severe constipation; then cream, or, better still, butter, is to be preferred.

In addition, carbohydrates are well borne in fever if well mixed in plenty of water; barley flour soups, or soups (preferably meat broths) with flour added, are also to be recommended. A certain amount of sugar may be introduced, in lemonade, milk, flour-and-milk soups, cocoa, cereals, and preserves. The quantity of sugar may be increased if, instead of cane sugar, Soxhlet's "Nährzucker" (without admixture) is used; this, however, constipates when used in large quantities. It is impossible to replace the loss of body albumins in fever by the administration of albumins in the form of eggs and meat, as these are absorbed only in small quantities even when given frequently and plentifully, and we must be satisfied with the quantity which can be given in the milk, and in starchy foods.

Nor is a preference for albuminous nourishment indicated when the appetite increases, inasmuch as a suitable combination of albumins, carbohydrates, and fats is best adapted to the physiological requirements. The nutritive value of carbohydrates in artificial preparations is indisputable. These baby foods may sometimes be of service in later childhood, as well as in infancy, although at this period it is better to give farinaceous foods which have been prepared in the kitchen. On the other hand, in infancy one can order malt extract or unfermented grape juice (alcohol free) occasionally. If offered in small quantity at the beginning of the meal, it also stimulates the appetite. Prepared foods which are rich in albumin (Puro, Sanatogen, Somatose) are hardly of much importance as nourishment, as they are used only in small quantities; but even they may be of value, either for their general effect

or for their ability to stimulate the appetite, especially the latter. The action of cod-liver oil, as well as other oils, is not very clear. They appear to act, however, by stimulating the appetite. All these preparations are to be discontinued as soon as a disinclination for them arises, owing to their interference with the proper taking of food.

Much less clear is the significance of those organic and inorganic substances which are not utilized in forming or replacing body tissues, but which are intended to do functional work; yet they should not be undervalued as nourishment. To prevent harming the patient through our lack of knowledge, we should allow sufficient play for choice and taste in the matter of food. For a long time, the theorist had difficulty in estimating the value of non-nutritious constituents which are present in more or less equally nutritious foods, *i.e.*, meat, eggs, vegetables, and fruit; and only the more recent investigations have shown, for instance, the importance of phosphorus combinations in building up (anabolism), and have demonstrated the value of fresh vegetables for the prevention of serious chronic diseases. But in spite of this, there are certain stimulants (spices, alcohol) which are always to be avoided in excess, and are to be withheld entirely from certain children.

As a rule, the number of meals should not exceed five, among which two full meals are sufficient. Where it is impossible to take enough food at one meal, a greater number of small meals should be given. Moreover, a dessert may be added a short time after the meal itself. Night feedings are rarely necessary. Under certain circumstances it is wise to offer frequent draughts of fluid to stimulate the patient, or to keep the mouth moist. This holds good, however, only in severe fevers and wasting diseases. It is only rarely, as in the constant vomiting of whooping-cough, that a greater number of meals must be considered. Nourishment should not have, as was formerly supposed, an increase of the weight for its sole aim, for under certain conditions and in certain diseases its action should be different.

Under the title of "The Exudative Diathesis," Czerny has described what, for the most part, has been understood as scrofula. For the past few years, he has advised in this condition the use of a vegetable diet with only small quantities of milk and meat. He prefers that eggs be avoided altogether, and gives no sugar, and but small quantities of butter. Leaving the question of exudative diathesis out of consideration temporarily, it is nevertheless true that there is a very close relation between certain skin diseases and the nourishment. In infantile eczema (in which the face is principally and most severely affected) the nourishment and particularly the quantity of milk taken, should be restricted. In the very common condition of urticaria infantum it is desirable to diminish the quantity of milk taken as well as the butter; while eggs and raw fruit should be entirely avoided. There should

be an increase of soups, vegetables, and starchy foods, but the number of meals should be decreased. A similar diet should be prescribed in intertrigo. In obesity, where it is found that a disproportion exists between weight and height, it is well to give a diet that is more filling than nutritious. It would, of course, be impossible and useless to attempt to influence excessive growth by a retrenchment of diet. In families with the gouty diathesis, children who are otherwise healthy but who show a preference for meats, had best be restricted to warm meat once daily, and perhaps some cold cuts once daily. A purely vegetable diet is hardly indicated in any of the chronic conditions. In neuropathic children, also, it is sufficient to withdraw meat broths containing the extractives which stimulate, and to allow meat once a day. Meat is on the same plane with other nervous irritants (alcohol, coffee, spices, confections). By avoiding these things, an over-stimulation of the nervous system will be prevented, and especially of the awakening sexual desires.

The avoidance of salt in the large quantity in which it is generally used diminishes thirst, and thereby the drinking of an excessive quantity of fluid. Simplicity in the preparation of food prevents an artificial stimulation of the appetite and the consequent overfeeding, while, on the other hand, a luxurious diet, at least for older children who are allowed to partake of these richer foods, leads to a decided stimulation of the activity of the digestive organs. A habit of simple tastes in food will find enjoyment in fruit, etc. This idea should be borne in mind, particularly where scrofula and the neuropathic habit are coexistent.

Care.—The care of the child is of just as much importance as is its nourishment. How often do we see children recover perfect health when both of these points have received attention, even when only the normal regime which is correct for the healthy child has been instituted! Aside from the wonderful healing qualities of natural food (*i.e.*, breast-milk) for infants, a mixed diet in older children will often immediately cure a disturbance in nutrition or digestion which has resisted all other methods of treatment. The extraordinary therapeutic value of proper nursing, like proper nourishment, may manifest itself in such an unexpected manner when employed in a case in which it has not previously been used, that the real importance of the treatment may not be realized.

In spite of the great value which hospital nursing possesses, particularly for small children, it should as a rule be looked upon only as a makeshift, of which too long use should not be made, notwithstanding the earlier statement. If a healthy infant requires almost the entire attention of a nurse, much more is this true of a sick one. With good training, a practical turn of mind and a gift for unbiassed observation, a nurse will succeed in obtaining results in private nursing by individualizing, which are impossible in the routine nursing of large numbers

in the hospital. Even though the child is not to be allowed to acquire bad habits, as it is apt to do in the leniency shown it during illness, it must nevertheless be remembered that long spells of crying and the free perspiration caused by the effort are harmful. In painful diseases of the bones (rachitis, infantile scorbutus, osteomyelitis), unnecessary disturbance of position would be cruel. On the other hand, in dyspnoea or a tendency to laryngismus stridulus, it is necessary to take the child up; and it is possible in some cases, as in earache, to ease the pain by simply carrying the child around.

Warming the bed is of importance not only in premature birth, but in many of the severe diseases as well. A particular position in bed may be required, owing to a tendency to vomit, to laryngismus stridulus, or in certain internal or surgical diseases. The bed itself should be hard in beginning rachitic kyphosis. Where there is a tendency to sweating, as in rachitis, the child should be only lightly covered with a woollen blanket.

Extreme cleanliness of body, the linen, and the clothing, will induce a more rapid recovery from many skin diseases (eczema), and will prevent complicating skin affections in acute diseases of the intestines, for instance, and in all protracted diseases accompanied by exhaustion and emaciation. For the cure of itching and vesicular eruptions, cleanliness of the hands should be given especial attention. In intestinal diseases, the mouth should be observed expectantly. In diseases of the mouth (thrush, aphthæ, etc.), a frequent and appropriate treatment is necessary, which, however, should not be such as to produce injury (Bednar's aphthæ).

The clothing, which in general should be suited to the season, should be neither too tight nor too heavy, and where there is a tendency to sweating, particularly in rheumatism, should be selected with a view to the ready evaporation of the sweat. Porous underwear made of stockinet, which should not be covered with impenetrable outer garments, is to be recommended. Under no circumstances should the neck and trunk be particularly closely enveloped, through the fear of repeated catarrhal attacks. Impermeable head coverings produce headache, but impermeable foot gear, unfortunately, often cannot be dispensed with, owing to the danger of wetting the feet.

Fresh, clean air for the sick room not only should be strenuously urged for its favorable effect upon all diseases of the respiratory passages, as well as for its importance in all chronic diseases; but it is absolutely required during such conditions as catarrh of the larger and smaller bronchi, and particularly in whooping-cough and pneumonia, and indirectly in rachitis, tetanoid spasms, and scrofulo-tuberculosis. To maintain the purity of the air, it is necessary to remove such furnishings as may create or retain dust, and to leave undisturbed the dust

which has already settled. The sick room is to be thoroughly ventilated day and night, either directly or indirectly (through an adjoining room), according to the weather, at the same time avoiding draughts. In conjunction with fresh air, bright daylight should be admitted into the room for the sake of its purifying and invigorating effects. The one exception to this rule is in inflammation of the conjunctivæ, as this occurs, for instance, in measles, in which case a deadening of the bright light is desirable. All these points in the nursing of the sick can only be touched upon here.

Psychotherapy.—In the care and treatment of a sick child at any age, the psychological effect is of the greatest importance. The inner life of the child is not sufficiently understood by physicians and nurses. So far as psychically normal children are concerned, to be successful it is necessary only to carry out all painless procedures without producing resistance on the part of the child. This can be accomplished by a gentle and friendly manner or by diverting the attention of the child, or through encouragement, which will cause the child to become trustful and docile. That many methods of treatment (*e.g.*, dietetic) are successful only under these circumstances requires no elaboration. Psychotherapy acts in a direct manner, since it awakens conscious perceptions and possibly fixes them through repetition, and by influencing unconscious perceptions by suggestion. Motor anomalies may be cited as examples of the former. Faulty positions of the body may be improved through training, and by means of suitable gymnastic exercises the improvement can be made permanent.

The influence of suggestion also has a broad field of activity, whereas hypnosis but rarely comes into play (*e.g.*, in the more severe cases of somnambulism). Many disorders may be made transitory in a similar manner, by preventing them, from the very beginning, from being strongly emphasized, and by thereby obviating the fixation of the disease conception. Even pronounced hysterical symptoms, such as uncontrollable vomiting, convulsions, etc., often disappear in a very short time if the patient is removed from his usual surroundings and his complaints are ignored, and in this manner the vicious circle of unconscious disease concepts is broken and repressed. Active suggestion often requires positive exhortation (by which the child is caused to concentrate his attention by gazing fixedly at the physician). An impressive manipulation which apparently or actually coöperates in the removal of the symptoms of disease is of greater assistance, such as medicaments, electric current, massage, passive and active exercises, etc. Suggestive therapy will be used the more, the more carefully the physician takes into account the psychological aspect of all diseases, of which chronic constipation, which has been found to have autosuggestion as a basis, may be taken as an example. And finally, the psychological influence

has its share in the successes of many varied methods of treatment (e.g., sojourn in the country, baths, sanatoria, etc.), without the physician or those around the child being always conscious of the fact. Association with children of the same age, the sense of new impressions, and change in routine and in the demands made upon it, improve the temper, give new impulse to the will, and bring about a change in the tone of all the functions which, if maintained within proper limits, may be of great therapeutic value.

Physical Treatment.—The physical methods of treatment in pediatrics are so highly thought of because of the simplicity with which they are carried out, and the certainty of their success, that there is even a tendency to ignore on their account the treatment of the etiological factors. The simplicity of these methods sometimes leads to forgetfulness of the fact that they may produce local or constitutional injury when used collectively, and particularly when frequently repeated. It is therefore impossible to regulate these procedures according to a fixed rule. The digestive tract, at its upper and lower portions, is most frequently subjected to mechanical treatment,—stomach lavage and gavage, procedures for emptying the bowels, and rectal feeding.

Lavage of the stomach may be used for removing harmful gastric contents in poisoning, or after the ingestion of indigestible food, and again for the removal of pathological contents (mucus, excess of hydrochloric acid, feces in stercoraceous vomiting). The most frequent clinical indication for this procedure is vomiting. But it would be an error to consider gastric lavage only as a method of assisting spontaneous evacuations of stomach contents, inasmuch as, by distending the walls of the stomach, it acts as a sedative and stops the vomiting. Hence it follows that an indication for lavage exists even when there can no longer be any possible suspicion of further retention of irritating contents in the stomach. It is, however, frequently possible in these cases to allay the vomiting by the much more simple procedure of administering a proper diet.

For gastric lavage a soft or semi-soft catheter should be used (Nélaton or silk-woven catheter), which should be connected by means of a short glass tube to a piece of rubber tubing about the length of the child; at the end of the rubber tubing there should be a funnel to facilitate the pouring in of the fluid for washing. The catheter should have the largest possible calibre (for infants, No. XV). The infant is then well wrapped (because the bowels are usually evacuated during the course of the lavage), the arms being included in the wrapping. The child is taken upon the lap of the nurse or the physician, and a second person pours the fluid, and allows it to flow in and out. If the physician holds the child, after having protected himself with an apron, he lays the child upon his lap, and with his right hand introduces the cath-

eter, which he holds in place, using his left hand to hold the infant, whose head must be toward the left and depressed. This method recommends itself, as in practice there is frequently a lack of assistance. In older children the nurse must hold the child, holding the legs between her knees, grasping the hands with her right hand, and with her left against the child's forehead pressing the head against her breast. The physician then introduces the tube, at the same time keeping the mouth open with a mouth gag, rubber wedge, or cork if necessary, which he steadies while another person allows the water to flow in and out. The water should contain a small quantity of table salt or Carlsbad salt, or, if it is desired to use a disinfectant, boracic acid; it should be lukewarm, and be slowly introduced, the height of the funnel regulating the rapidity of the flow; it is emptied according to the principles of Heber. This should be repeated until the water returns clear. As it is possible that stomach contents may be retained out of reach of the flow of water, it is necessary to change the direction of the tube and to withdraw it if it comes in contact with the wall of the stomach; if blood or mucous membrane appears in the water, for safety, the tube should be at once withdrawn, and the procedure be discontinued.

Instead of this method for washing the stomach of the infant, that recommended by Escherich may be employed. It consists of a graduated bottle with a wide neck, having a capacity of about 400 c.c. (1 pint). This is closed with a rubber cork, having two small openings. Through one of these passes a glass tube which reaches to the bottom of the bottle, and through the other passes a shorter one; to the former is attached the stomach tube, and to the latter a short piece of rubber tubing. After introducing the stomach tube, the contents of the stomach may be evacuated into the bottle if desired, and then be subjected to examination. The cork is then withdrawn from the bottle, which is replaced by one containing fluid for washing the stomach. By blowing through the tube connected with the short glass rod, the fluid is forced into the stomach and is returned into the bottle by the pressure of the abdominal wall. This procedure is repeated with changed cleansing fluid as often as necessary. This method makes it possible to judge more accurately the pressure and the quantity than can be done in using the tube and funnel.

The same kind of catheter and funnel, but with a shorter rubber tubing, is employed for the introduction of fluid nourishment into the stomach. The funnel, tubing, and catheter are filled with the food; the tubing is then pinched while the catheter is being introduced. When the tendency to choking, after the introduction, has ceased, the fluid is allowed to flow as slowly as possibly, to approximate the normal intake of food, interrupting the flow whenever coughing or choking occurs. When sufficient nourishment has been introduced, the tubing is again

pinched and the catheter is withdrawn as quickly as possible. In many cases where food is refused owing to weakness, it is possible, at least with small children, by carrying out this method of feeding three to five times daily, to gain time for several days, until nourishment is taken naturally. Sometimes, however, the procedure is attended by failure, the contents which were introduced being expelled immediately, or within a short time. It is particularly in such cases, where the stomach is unable to retain anything, that *rectal feeding* should be considered. Here it is a question of introducing substances which will be readily absorbed; *i.e.*, salt, sugar, and alcohol in aqueous solution, beside fat, but, above all, water itself. As a rule, it is possible to give a nutrient enema only four to five times in the 24 hours, for a few days. It is hardly to be expected that a sufficient quantity of nourishment can be introduced into the body in this way for any great period. The quantity ranges from ten to thirty Gm. (3-8 drams) in infants, up to one hundred Gm. (28 drams) in older children, and only rarely is larger than this. A rectal irrigation must be given once daily for cleansing the bowel, after which the bowel should be allowed to rest at least an hour before introducing the nutrient enema.

For small enemata—about ten Gm. (3 drams)—a small rubber bulb is used, or, better still, a small piston syringe with a tip sufficiently long. For larger children use an irrigator with a longer semi-soft tip, the tip being well lubricated. Avoiding the entrance of air, the lukewarm fluid is allowed to flow slowly into the rectum. It is best to take milk, to which a little salt and sugar have been added and into which an egg may be stirred (one egg to four ounces of milk). As the fluid is generally the essential ingredient, the white of egg can be omitted, should it cause irritation of the rectum through putrefaction. In its stead alcohol (cognac, etc.) can readily be used if the occasion demands. Particularly in young children, irritation of the mucous membrane, with discharge of mucus and tenesmus, soon occurs, or the nutrient enema may be soon expelled, in which case tincture of opium may be carefully added. The indications for rectal feeding, therefore, should be held within narrow limits; *i.e.*, in the uncontrollable vomiting of small children, or the temporary closure of the upper portion of the gastro-intestinal tract, better results usually being obtained by other methods.

The mechanical emptying of the rectum is the most frequent procedure employed in the nursery, either with rubber bulb syringes (less frequently with piston syringes), or else with the fountain syringe. The fluid is permitted to enter while the child is in the ordinary position on the side, or upon the back with raised buttocks, or even, with older children, in the knee elbow position. This softens the mass in the bowel, and, assisted by the reflex irritation of the abdominal and intestinal muscles, should produce an evacuation. An indifferent fluid is usually

all that is necessary (chamomile tea, or six-tenths per cent. salt solution). Soap water, which is so popular, is more harmful, and is therefore to be avoided in small children; while castor oil (with egg or a little starch), or, if need be, a decoction of senna leaves (one dessertspoonful to one quart of water) floated upon the water works powerfully and with little irritation. The quantity of the enema ranges between 30 and 50 Gm. for infants and 100 and 250 Gm. for larger children. Smaller quantities (30 to 100 Gm.) are required with oil enemata, which should be retained until the hardened fecal masses have become softened, when after a few hours they will be evacuated spontaneously, for which reason the bed should be well protected. Glycerin enemata occupy a special place, because of the small quantity required (one teaspoonful to one dessertspoonful in equal or double the quantity of water to prevent irritation of the bowel); they are introduced by means of the small rubber bulb or piston syringe, not by means of an irrigator.

Aside from the injuries which may result from the use of hard tips in giving enemata, particularly where the child strains, the ampulla becomes dilated through their constant use, and, even where this does not occur, they lose their effect. If the bowel must be emptied artificially there should at least be a change of method from time to time. But certainly, in most cases, where there is no congenital dilatation of the colon, it should be possible to overcome constipation by dietetic or mechanical methods; *i.e.*, by change in the character of the food, by more exercise, or by abdominal massage. There are a certain number of cases of chronic constipation which fail to respond to ordinary methods and which are psychical in origin; they should therefore be treated by suggestion.

That in the temporary constipation which occurs during acute febrile diseases, an enema is the simplest and quickest procedure, there can be no question; but here also a fixed rule is to be avoided. In contrast with those injections that are employed simply to remove feces, larger injections should be used in acute catarrhal colitis and less often in chronic disturbances of the colon, for the removal of poisonous and irritating matter, thus attacking the seat of the cause of the disease. For local effect a medicament may be put into the first or subsequent injections (tannin, one per cent.; liquor alumini acetatis (P. G.), one per cent.; liquor plumbi subacetatis, one per cent.,—the latter to be used only once or twice). These injections should not be retained, like those given for moving the bowels, but should be given in a constant stream which is at once allowed to escape; if at the end, however, some of the solution is retained and absorbed, it is of benefit in most instances. In intestinal invagination, high and copious injections are of use only if the invagination enters the lowest portion of the colon.

Finally, we have to allude to medicinal enemata (*e.g.*, decoction of

garlic for the removal of intestinal parasites); the smaller enemata (starch solution) for quieting the inflamed mucous membrane; and the stimulating enemata (vinegar, 1 to 4-5).

Of the various methods of treatment by introducing substances through the skin into the tissues, only the subject of *subcutaneous infusion* deserves a few words at this point, as the use of drugs by hypodermic injection is sufficiently well known. Subcutaneous infusion may be very useful in the first place, in replenishing the tissue fluids lost through exhausting vomiting and diarrhœa or severe hæmorrhage; and, in the second place, in septic and toxic conditions, by causing greater drainage through the tissues. It is only necessary to use it opportunely and with sufficient frequency—even two or three times daily. In pediatric practice, the principal indication for this procedure is in cases of diarrhœa and vomiting, when the absorptive powers of the intestines have been lost, and the decrease of the normal tension of the skin indicates the danger of the drying out of the tissues. Instead of all the complicated solutions, it is only necessary to prepare a salt solution (six or eight Gm.—about one heaping teaspoonful—of table salt to one litre—one quart—of water), and to boil it for ten minutes. For injecting the fluid, one of the larger syringes is used, such as is commonly employed for the injection of serum, and which is usually at hand. The injection is accomplished by refilling the cylinder repeatedly without removing the needle from the tissues. The large syringes which are connected with a cannula by means of a rubber tubing are much better. It is also possible to introduce fluid under the skin by using a length of rubber tubing which has been carefully boiled, one end being attached to a needle and the other to a funnel. Only 30 to 50 c.c. (8 to 14 drams) in infants, and 50 to 100 c.c. (14 to 28 drams) in older children, can be introduced at one point. If necessary, however, the infusion may be made at a number of points. During the infusion the needle is slowly advanced and must always have its point free in the subcutaneous tissue. The preferred location for the infusion is the region below the clavicle, or the side of the abdomen. Local reaction can readily be prevented by the sterilization of the instrument and the skin, and by closure of the puncture with plaster, or cotton and collodion.

Bleeding is also occasionally indicated in childhood. Its action is sometimes very rapid in uræmia; but less often will it help to overcome the danger of death in conditions of extreme cyanosis (*e.g.*, pneumonia, cardiac weakness) by relieving the circulation. As a rule, however, it should be practiced only in the latter half of childhood, 80 to 120 Gm. (3 to 4 ounces) of blood being drawn. An elastic band or a wet gauze bandage is wound rather tightly around the pendant arm, so that the peripheral veins become distended. After making a small incision through the disinfected skin, a vein in the bend of the elbow is opened

for a distance of about $\frac{1}{2}$ cm. in the direction of the course of the vessel, and the blood is allowed to flow into a receptacle whose capacity is approximately known. After removal of the bandage the bleeding ceases spontaneously, so that only a small antiseptic compress is required at the point of incision. Occasionally, it is possible to puncture a fully distended vein through the skin by means of a good sized cannula.

Blood-letting by means of wet cups, in the vicinity of a diseased organ, is also to be chosen only in the case of older children (particularly in inflammation of the lungs and pleura); whereas dry cupping may be employed in younger children. A wet cup draws up about 3 to 5 Gm. (45 to 75 minims) of blood. Each cup is taken directly out of warm water and its opening is held over an alcohol flame, and then, before it has become too hot, it is quickly and firmly pressed upon the skin. To remove the cup, the skin at its margin is pressed down, when the entrance of air into the cup releases it. Instead of wet cups, leeches may be used. Puncture of the spinal canal, which is of value not only for diagnostic but for therapeutic purposes, will be considered elsewhere.

Hydrotherapy (including balneotherapy).—Most of the diseases of childhood are favorably influenced by the use of water applied in any way. Only in certain diseases of the newborn, and in some skin and surgical affections, are there any contraindications. Like any other remedy, when misused, water may become harmful, and it is often used only to supplement some other form of treatment. If, therefore, hydrotherapy cannot be considered as a separate method of treatment, as distinguished from the rest of the subject of therapeutics, it nevertheless enjoys the particular advantage of careful scientific elaboration and a readily controlled action rendering possible the most delicate graduations and changes in the application of its various procedures. This advantage, however, is of value only where the action of every prescription is watched by the physician himself, and the procedure is modified according to necessity and to the result obtained, which latter depends upon the individuality of the patient and the course of the disease.

Water, acting upon the body surface, affects the nerves, vessels, and tissues. The behavior of the blood vessels, influenced directly or reflexly through the nervous system, is particularly important in so far as the primary effect of the treatment is followed by a secondary action (really in a direction opposite to that of the first), which affects their state of contraction. Most of the procedures produce their best results only when this opposite condition, *the reaction*, takes place promptly and markedly. During the application of the water treatment, aside from local changes in the amount of blood in the vessels, there will also be an opposite action (revulsion) upon the distribution of the blood in

the underlying tissues or in the tissues of other parts of the body. It is therefore, possible, directly or indirectly, to influence the quantity of blood in a given area, increasing or diminishing it, and thereby changing its nutrition and function. Procedures which include a considerable portion of the body surface, acting through the circulation, have a more powerful influence, even affecting general metabolism.

Beside the consequent circulatory changes in the skin or other organs (*e.g.*, the muscles and the brain), hydriatic measures affect the nervous system directly, by the irritation of nerves arising in the skin, so that, whether in these or other ways, the psychical functions and the innervation of the different systems of organs may be strongly influenced.

It is necessary to dwell only briefly upon the subject of circumscribed cooling and heating of the skin. In using compresses, they must be changed so frequently (a detail which is often neglected) that instead of an alternation of heat and cold, the temperature will be constant. Prolonged cooling is made more effective when an ice bag of sufficiently large capacity and surface is placed upon the wet compress. Heat may be fairly constantly applied by means of cereal poultices (cataplasms) composed of oatmeal or flaxseed, which may be kept warm in a poultice warmer. Temperatures of any desired degree may be conveniently employed by means of coils made of aluminum or metal tubing, coiled and sewed upon flannel and laid upon the part, water of the desired temperature being permitted to flow through the apparatus from a larger vessel situated above the level of the part, to a vessel placed upon the floor. Aside from surgical diseases, the prolonged action of cold may be employed in all acute inflammations; upon the head in inflammation of the brain or congestion from any cause, which produces headache; upon the chest in inflammation of the lungs, but especially in inflammation of the pleura; over the heart in inflammation of that organ, or when, as in prolonged fevers, cardiac failure is threatened from being overtaxed; and, finally, upon the abdomen in circumscribed or general inflammation. If pressure cannot be tolerated, as in peritonitis, compresses are to be preferred, or the ice bag is suspended in such a manner as to just touch the surface. If motion is to be avoided, and if pressure does not disturb the patient, the metal coils are perhaps preferable. When wet compresses are used upon the rest of the body, they are to be covered with some impervious material, in order to prevent the unpleasant wetting which would otherwise occur.

The difference between cooling and hydropathic compresses is often misunderstood. In the one case, the compresses are so frequently changed that the skin remains constantly cool and anæmic; in the other, it is allowed to remain for a time upon the part, so that finally it acquires the temperature of the skin, which thus becomes congested; and not only does a subjective sense of pleasant warmth follow the fleet-

ing sensation of cold, but the moisture of the compress actually approximates more and more the temperature of the blood. These hydropathic compresses, which attract the blood to the surface of the body, instead of driving it from the skin to the diseased organs, as the cold compresses probably do, are exceedingly efficient in diminishing pain at least, beside which they frequently improve the local disease.

Hydropathic compresses are applied, after first anointing the skin when it is sensitive, by taking a linen or raw-silk cloth, folding it into layers of sufficient thickness, immersing it in lukewarm water, wringing it out until it no longer drips, and covering it with a woollen cloth which overlaps it liberally, or with a woollen binder held in place by pins or a bandage the compress to be changed every two or three hours, or, under certain circumstances (*e.g.*, at night), at longer intervals. Should it become necessary to apply such a compress to the chest of a small child, for instance, it is best to place it all prepared upon the dressing table or bed, the woollen cloth underneath and the wet one upon that, and to lay the naked child upon it, so that the compress can be speedily wrapped about it. To change, a second compress is held in readiness. To avoid inflaming the tender skin of the child, new cloths should be used daily wherever possible, those which have been used being washed before decomposition of the fabric, with its accompanying sweetish odor, has taken place, as this is capable of irritating the skin. For very young children, a chest compress may be prepared in the following manner: a wet undershirt is put upon the child, over this a tricot shirt not too thin, and a binder is wound around all. If it is desired to cover the upper portion of the lungs with a compress, a cross bandage is necessary. This is applied by even turns of a binder made of handkerchiefs or pieces of linen folded together, first one turn wet, then one turn dry. These are best covered with a woollen binder or a woollen shirt. For example, one starts from the left side of the chest anteriorly, and below, over the right shoulder to the back, then to the left axilla, passing forward under it and horizontally across the chest to the back; from there over the left shoulder, across the chest anteriorly to the right axilla, and once more horizontally around the chest. The full pack is less frequently used than is the body compress, and differs from the latter in that it includes the extremities. In applying a full pack, the dry outer cloth should be somewhat larger than the wet one, and may be made of woollen blanketting, or towelling; the inner wet compress may be of folded sheeting. These are spread out, the wet one uppermost, and arranged smoothly and evenly, ready for the child to be placed upon it in such a manner that the wet compress extends from the neck to beyond the heels. The child is then quickly enveloped in the compress, which is pushed down between the legs and thighs, and the arms and body, and is brought up over the feet; the dry blanket or towelling is wrapped around the body

in the same way and is fastened by tucking it in at the neck and wrapping it around the feet. The bed cover is then placed over all.

Compresses are also used to influence the distribution of the blood in a definite manner. A leg pack, a compress around the abdomen or the body, or finally, a full pack, tends to act as a sedative or a hypnotic by drawing the blood away from the head. The full and body packs are particularly important, since they have the power on the one hand of reducing temperature, and on the other of producing sweating, and even an increase in temperature. The cooling action depends upon the temperature of the water used, the degree of saturation of the compress, and the frequency with which it is changed; and, indirectly, upon the extent of the body surface, the condition of superficial circulation, and the height and persistence of the fever. In small children a body pack is usually sufficient, the water being lukewarm, 24° to 20° C. (75° to 68° F.). In the case of larger children, it is used at room temperature in winter, and in summer as it runs from the faucet, 20° to 12° C. (68° to $53\frac{1}{2}^{\circ}$ F.). The compress is changed about every twenty minutes, but usually every half hour or even every hour. Having obtained a satisfactory result, the compress may be left on, or it may be taken off, the body being thoroughly dried and then warmly wrapped.

If warmth or sweating is desired, the full pack is to be preferred, at least for larger children. If sweating is desired, tepid or even warmer water is used, the child is very warmly covered, hot water bags or bottles are placed at the feet and sides, and hot fluids (milk, elder flower, and lime tree flower tea, lemonade) are given to drink. A cloth is placed under the chin and the perspiration is wiped away from the face as soon as it forms. In conditions of weakness, particularly in babies, it is necessary to raise the temperature of the body. To bring this about, two compresses are prepared at the same time; one is dipped in water so hot that it can just be wrung out, and the child is quickly wrapped up in it. In ten minutes it is removed and the other is used in the same manner, and after perhaps six changes the temperature will have risen sufficiently to place the child, temporarily, in its warm bed. To prevent the raising of the temperature beyond the desired point, constant use of the thermometer is necessary.

To obtain proper results from the use of the various compresses, it is necessary that the skin shall become congested. Failure to react is especially likely to occur in the use of cold compresses, from the incidence of a sudden rise in temperature (as with a chill), or when the compress used is too cold. In such cases, it is well to rub the skin with the compress before it is applied, or to give a rubbing with alcohol, French brandy, camphor, or even oil of mustard, or perhaps a washing with hot water. When the pack induces free perspiration, the water treatment may be concluded with a cool sponge bath, which causes the vessels

of the skin to contract again. This is done by using a cloth dipped into water at the temperature of the room, washing and then drying in turn, arms, legs, chest, and back, keeping all of the body covered except the part being washed. If, on the contrary, the sweating is to continue after the pack has been removed, the body is but lightly dried and the child is allowed to remain in bed warmly covered. In many individuals sweating is produced only after repetition of the procedures properly performed.

The same effect upon the temperature of the body can be produced by baths as well as by compresses, or by a combination of the two. A bath is not quite so easily managed for a child as is a compress; but it is much more easy than in the case of an adult, and it is therefore of extended therapeutic application. The only contraindications are inflammation of the skin, particularly of a moist type, great bodily weakness, and fear of the water. But even when these are present, suitable additions (*e.g.*, bran), appropriate temperatures, or especially gentle treatment (*e.g.*, slow immersion by means of a sheet), may make it possible to use the procedure. In a small bath tub which rests upon a stool and holds about 10 litres (three to five bucketfuls), the infant is held suspended upon the left hand and forearm, while with the right hand it is washed and gently rubbed.

When, in children suffering from high fever, no redness of the skin occurs under this treatment, it becomes necessary to produce much stronger friction with the hand, or a rough cloth, or even a friction glove. Since in the newborn the normal bath temperature, 35° C. (95° F.), when used for several hours is capable of reducing the temperature of the child, it is important not to make the bath too cool (in delicate infants 33° to 32° C. (91°–90° F.) should be the minimum), and even for larger children, a lower temperature than 30° C. (86° F.) should not be used for reducing fever. The rapid appearance of redness of the skin is necessary to indicate the benefit and success of the procedure, and is often obtained much more readily by using a bath of higher temperature for a longer time (ten or even fifteen minutes), than by using a colder bath for a shorter time, say three to five minutes. It is often found best to begin the bath at the usual bath temperature, 35° C. (95° F.) and gradually to lower the temperature by allowing cooler water to flow in at the foot of the tub. To give a hot bath, it is started at 35° C. (95° F.) and is raised to a temperature of 38° C. (100° F.), or even to 40° C. (104° F.), by the addition of hot water; or it may be started at a higher temperature. The bath lasts from five to fifteen minutes, and the child, after being dried, is laid in the warm bed.

The hot bath was formerly given only to produce sweating, but it may also be used as a revulsive or a stimulant. Thus, hot baths (up to 40° C.; 104° F.) have proved their value in the afebrile stage of

epidemic cerebrospinal meningitis, and these may be of considerable value in the most varied conditions of weakness, and may even be given to infants. In flabby, scrofulous, or anæmic children, who are not high-strung, warm or even hot baths may be given according to some definite plan two or three times weekly for a period of four or five weeks. If it is desired to prolong the action, the patient is kept in bed for several hours thereafter. If there is danger of making the patient too susceptible (tender), or if greater stimulation of the nervous system is desired, a quick cold douche is given by pouring water upon the nape of the neck from a pitcher or a sprinkling can. A cold douche is an excellent procedure after a cooling bath, increasing its stimulating effect and tending to cause a deeper respiration. When the bath is but the initial step in a combined water treatment, this douche must not be employed, as one may, for instance, follow immediately with a pack. A cooling bath is but rarely supplemented by a cold pack, whereas a hot bath is very frequently used as a preparation for the full pack, which is to induce sweating.

Between the hot and the cold full bath lie the "indifferent" full baths at 34° to 35° C. (93° to 95° F.), which act as a sedative or hypnotic, especially when of long duration (about one-half hour). Spastic conditions, even when of organic origin (as in cerebral palsy), are relieved, so that passive movements may be made more readily while the patient is in the bath. Mental excitement is allayed, mental strain is relieved, and sleep, which has been absent, occurs more readily after the bath.

A similar but less marked action is obtained by means of the sitz bath. The lower part of the body and the upper portion of the thighs are immersed in a wooden tub, or an ordinary hip bath tub, or, in the case of very small children, in a very deep bowl, the upper portion of the body being protected from exposure by a large bath towel. The temperature should be between 33° to 30° C. (91° to 86° F.) and the duration from three to ten minutes. As the reaction produces a congestion of the genital organs, this procedure should be avoided just before bed time. Sitz baths are of value in all conditions of nervous irritability (chorea, cephalalgia), but particularly of the genito-urinary system (enuresis, onanism).

In considering the half bath, we come to the measures which act as nerve stimulants, of which measures others will be mentioned. Of the previously described procedures, the cold full bath should be included in this class, excepting that it is not, as a rule, used for children, at least so far as a tub bath is concerned. On the whole, it has been found that none of the procedures which have a markedly stimulating effect upon the nervous system may be used too vigorously, or for too long a period. By proper application and with due regard for individual susceptibility, the nervous system may be refreshed and strengthened;

whereas, on the other hand, much harm may be done by overstimulation where this susceptibility is not considered.

The half bath reaches to the umbilicus of a child when sitting in the tub. In the case of smaller children, the difference between a half and a full bath often disappears. Only at the end of childhood may the half bath be given as cold as is generally advised, *i.e.*, 28° C. (82° F.), with a douche at 15° to 20° C. (59° to 68° F.), and even then a temperature somewhat higher, 30° to 32° C. (86° to 89° F.), is to be preferred. The arms and legs are rubbed with the water of the bath, which is at the same time poured over the chest and back, and toward the close the colder water is poured over the patient, the pitcher or other vessel being held as close to the skin as possible. Contrary to the usual advice to continue the procedure for ten to fifteen minutes, it will be found that three to five minutes will prove sufficient.

Another procedure which is very stimulating is the cold rubbing, which has the advantage of being readily given in the bedroom without much preparation. A bath towel which is slightly longer than the patient and wide enough to be wound twice around the body is dipped into a pail containing cold water, at first about 30° C. (86° F.) then gradually at room temperature, and finally as it runs from the tap or spring. After having been wrung out (as much as possible at the beginning of the treatment, later on perhaps not so much), the towel is wound round the child (who must be standing) as quickly as possible, passing it under the outstretched arms at the first turn and over the arms, which have been dropped to the sides, at the second turn, so that the towel will then be smoothly applied to the body from the neck down. Then with the flat of the hand, one anteriorly and the other posteriorly, the body is rubbed with long strokes through the towel, until the pack becomes warm. It is then quickly thrown off, and is replaced with a dry towel, which is used to dry the patient. After this the feet and legs of the patient, who is now seated, should be rubbed energetically and dried. A wet compress to the head during this procedure is unnecessary in the case of a child. Only weak and anæmic patients should be permitted to go to bed after this; as a rule the child is quickly dressed, and sent out into the open to exercise freely.

The cold sponge bath is even more simple, and may be considered as one of the hygienic measures in health. The cold sponge bath which must be given in bed to very delicate or sick children has already been described upon page 275. Otherwise, the child is made to stand in a flat vessel (of rubber if it is to be transported) or bath tub, and washed superficially all over the body with a cloth dipped in cold water but not wrung out; or the water is squeezed out of a large sponge and allowed to trickle over the body. This may be followed by pouring water from a pitcher or watering pot. The body is then dried.

The simplest procedure, but one which has a distinct power as a nerve stimulant, is the spinal douche. The child sits erect upon the edge of a bath tub or wooden tub, and cold water is allowed to fall upon the median line of the back for not more than one minute.

The shower bath is considered in this place only as a spray. The size of the drops must be small, the pressure but slight, and the duration short, from less than one minute to about two minutes. A lukewarm shower bath is about equal to a lukewarm bath. If the water is gradually cooled, the action is increased, because the stimulation from the cold and the mechanical stimulation are successfully combined. The Scotch douche of alternately hot and cold water, may be ordered for children only with great care; for instance, alternately 38° C. (100° F.), 30° C. (86° F.), 35° C. (94° F.), 28° C. (82° F.); and the following day with a greater difference—36° C. (97° F.), 22° C. (72° F.), 36° C. (97° F.), 22° C. (72° F.), or, in the case of nervous children, only in the sequence of 38° C. (100° F.), 30° C. (86° F.), 38° C. (100° F.), and 30° C. (86° F.), proceeding to 38° C. (100° F.), 26° C. (79° F.), 38° C. (100° F.), and 26° C. (79° F.). The cold showers may often be used with great advantage as the final step of a combined hydriatic treatment.

In the same manner a hot pack or bath may be followed by a tepid or cold bath. At home, by means of the shower, one can quickly cool the surface of the body, in the simplest possible manner thereby restoring tone to the blood vessels. All hydriatic measures intended to stimulate the nervous system accomplish this object only when the patient experiences thereafter a pleasant sensation of warmth, increase of energy and of bodily strength. This sensation of warmth must be maintained as long as possible by active though gentle exercise, in the fresh air if possible.

Hydriatic measures should always be looked upon in the same light as medicine, and their application should therefore not be left to the discretion of the uninitiated, and should not be kept up longer than from four to eight weeks, without interruption. The action of the water may be changed by the addition of some chemical introduced artificially, or by the use of natural water in which it already exists. The difference between the medicated baths and the natural mineral waters is not marked, inasmuch as the latter may in many instances be imitated artificially. The action of warm or hot full baths, or hot packs, to which aromatics have been added is particularly beneficial, their sedative action being increased. Infusions of chamomile, sweet flag, and of various aromatic herbs are used for this purpose; or extract of pine needles may be added to the bath. On the other hand, the addition of irritants to the bath is of value in deep-seated inflammations (*e.g.*, pneumonia), by acting as revulsives upon the skin, or by acting as a stimulant to the

nervous system in conditions of weakness. Mustard packs and the mustard baths (four to five tablespoonfuls of powdered mustard enclosed in a small linen bag) are the most useful of this type. It is unnecessary to discuss the local application of mustard paste and mustard plaster at this time. It is impossible to make use of mustard when the skin is very delicate or in a state of irritation. Astringent baths are given when moist eruptions of the skin exist, tannin, or, better still, decoction of oak bark, being added to the water. (A cup of pulverized bark is added to one litre of cold water and is boiled for one-half to three-quarters of an hour, after which the decoction is strained and added to the bath.) The irritation which water is apt to produce upon recently inflamed skin may be allayed by adding a decoction of white bran or malt to the water.

Sulphur baths are to be used in itching skin eruptions unaccompanied by moisture. 25 to 50 Gm. (1 to 2 ounces) of sulphurated potassa (100 Gm. (3½ ounces) for larger children) is dissolved in water and is added to the bath, after which the skin is thoroughly anointed with some neutral salve. Among the natural sulphur baths which are prescribed may be mentioned those of Heustrich, Leuk, Stachelberg, Kreuth, Nenndorf, Schinznach, and Landeck.

Baths which are both astringent and disinfectant are prepared with permanganate of potash; disinfectant baths, with bichloride of mercury, (1-20,000). But when the skin is not intact, the mercury must be administered internally and the baths be omitted. These baths are used in abscesses of the skin, and the bichloride baths are employed also in syphilis with localization upon the surface of the skin.

Instead of medicated baths, one may use other methods of skin medication, such as rubbing in tar preparations or medicated soaps, and following this with an ordinary bath, by which the skin is finally cleansed.

When the nervous system, the circulatory organs, or the general state of health is to be influenced the natural water baths are to be given in preference to those artificially prepared, because these baths when given at the watering places, according to tried rules, are more easily and satisfactorily administered, which assists in a marked degree in obtaining the desired result. In making a choice of baths, or of health resorts, it is not only necessary to determine the action of the waters, but also to take into consideration the entire condition of the watering place; indeed, one must bear in mind the many points which are of importance especially for children. The writer is personally acquainted with most of these resorts, but it is impossible here to go into the subject in detail.

Despite the fact that the so-called indifferent hot springs apparently depend for the reaction upon the degree and constancy of their temperature, the latter being obtained by means of a continual ebb and

flow of the water, they should be classed here with the natural baths. The invigorating and strengthening action of the warm springs makes them particularly valuable for all weakly children, and especially for those suffering from neurasthenic disorders, while the hot springs are especially highly esteemed in rheumatic disorders. Of the warm springs may be mentioned those of Vöslau near Vienna, Johannisbad, Baden-Baden, Badenweiler, and Schlangenbad; and of the hot springs, Teplitz and Warmbrunn.

The physical action of the different baths is aided by the presence of carbonic acid and minerals. The nature of the mineral present can hardly be considered of much importance, however, and if steel baths, for instance, are especially renowned for their effect in anæmia and chlorosis, it must not be forgotten that in these places water containing iron is internally administered at the same time. As a matter of fact those baths should be considered as a whole, whose action, like that of the cold water treatment, stimulates the nervous system, through which the vascular system is powerfully affected, differing only in this, that there is almost an entire absence of shock, and that the action is more prolonged.

The heavy salt solutions, such as those which manifest themselves by depositing innumerable bubbles of carbonic acid gas upon the surface of the skin, may produce such an unfavorable influence not only upon the tonicity of the peripheral vessels, but also upon the heart action, that the immediate reaction may continue for hours, and the indirect effect perhaps for days, while it may be even longer before the normal physiological balance is restored. For this reason it is necessary to find out in every case the temperature and duration of the bath which each individual can tolerate. This is to be judged by the subjective sensations, and objectively by the tension and frequency of the pulse. After the bath, the patient should be made to rest in bed for one or more hours, and another bath be given only when the reaction from the previous one has disappeared. An interval of one or two days must usually be allowed, during which the course of treatment demands bodily rest and a more liberal but simple diet. After the course of baths has terminated, these precautions in regard to rest and diet are continued as an aftercure.

Owing to the great fluctuation occurring in the distribution of the blood (after temporary contraction, dilatation of the peripheral vessels), and the accompanying fluctuations which appear in the innervation of the heart and blood vessels, constitutional diseases, such as rickets, scrofulo-tuberculosis, and anæmia, are distinctly influenced, and old inflammatory processes of the internal organs are cured. The vicarious action of the skin as a derivative also acts favorably upon inflammations of the kidneys. The baths when they are properly used, present

an exceedingly effective exercise cure for the circulatory system, and particularly for the function of the heart, which it may improve, if this has not already been too seriously affected. Whether the action upon the skin, which in turn reflexly influences the innervation of the blood vessels, is due more to the solution of salts or to the contained carbonic acid, is not definitely known; the action of the salt baths, the carbonic acid salt and mineral baths, and the simple carbonic acid baths differ probably only in degree.

In considering the mineral baths, we are dealing essentially with sodium chloride, which exists more or less in combination with other salts; these salts collectively are given as salt or brine baths. The use of the salt baths at home has increased extraordinarily; but when given without proper consideration these are often misapplied, as when they are administered for months, even though, as is the rule, they are given in weak solutions. The salt content should average about two per cent.; in the case of delicate young children, one and one-half per cent.; in older children, three per cent. During the course of the treatment the strength of the solution reaches even as high as five per cent. The occurrence of a greater tranquillity should be increased, without allowing this to reach the degree of drowsiness which, however, may be taken as an index of its action. Of the natural brine baths may be mentioned Ischl (comfortable and correspondingly expensive), Gmunden, Aussee, Kreuznach, Reichenhall, and Harzburg; and for cures to be undertaken in the spring and fall, Bex-les-Salines and Kalberg, which have mild sea climates; Elmen and Salzungen are sunny; and Kösen, Sulza, Suderode, and Orb may be recommended for younger children.

In considering the brine baths containing carbonic acid, it does not matter whether they are warm when they flow from the ground or are heated afterwards. Among these are Soden, Homburg, and Oeynhausen. The baths at Rothenfelde are simpler. Nauheim and Kissingen are to be preferred in cardiac affections.

The mineral baths and the iron baths (Stahlbäder) containing carbonic acid should be considered here. In the former the sodium chloride is displaced by other salts (alkaline-mineral and alkaline-saline springs). In all these the salt content is relatively low, so that the pressure of solutions, even when of a somewhat higher specific gravity, is neutralized by the action of the carbonic acid gas, and these baths consequently have a still milder action than the brine baths which contain carbonic acid. They may be ordered in conditions of general weakness, and irritability, in combination with the other factors which go to make up the advantages of a bath treatment (Badekur).

Among the alkaline-saline baths are Franzensbad, Elster, Marienbad, and (only for larger children) Tarasp. Of the steel baths there are Liebenstein, Reinerz, Schwalbach, Pyrmont, Flinsberg (about 3000 feet

high), and St. Moritz. The latter has the greatest altitude, and should therefore be recommended only for grown children; it is expensive.

The carbonic acid baths may be prepared at home with or without the addition of sodium chloride. The carbonic acid is liberated from carbonates by the action of sulphuric or acetic acid; zinc bath tubs, if not enamelled, are easily destroyed thereby.

In this place too are to be mentioned the peat and mud baths (Moor- und Schlamm-bäder), which depend for their action upon the constant temperature and general pressure over the surface of the body. As full baths they would scarcely admit of application to children; but, on the other hand, they may be exceedingly useful when locally applied at the site of chronic inflammations, to absorb old exudates (*e.g.*, in appendicitis after it has run its course, in tuberculous peritonitis, in tuberculous pleurisy, or in old rheumatic inflammations). The local pack is continued daily for weeks, an hour or so at a sitting, while at the same time the rest of the body is tucked into warm blankets. They may even be applied at home, *e.g.*, with the Fango mud; but it is better to employ them in institutions such as exist in the larger cities (*e.g.*, Berlin), or, best of all, in the watering places where they are used in connection with a cure. The following are a few of the many mud baths (Moorbädern): Franzensbad, Elster, Marienbad, Königswart, Pyrmont, Kissingen, Reinerz, Nenndorf.

Packing in hot sand is not to be considered as a bath in the full sense of the word; but its action, when it is used in chronic rheumatism, is related to that of the mud baths.

Steam and hot air baths are not used for children. By way of exception, however, may be mentioned electric light baths when employed to induce sweating, and sunlight baths used systematically by exposure of the body to the sun's rays,—the head being protected the while, and one of the cold water procedures being used in completion. These baths should be administered only very carefully.

Mechanotherapy.—The physical methods of treatment heretofore considered, had for their principal sphere of action the skin and mucous membrane. The methods which affect the motor apparatus will now be considered. Their importance depends upon the fact that they influence the circulation, heat production, and metabolism, the proper performance of these functions being responsible for the feeling of well being, and the general tone. A complete rest of the motor apparatus will diminish the heat production while by enveloping the body the dispersion of the heat already produced will be retarded, thus effecting the same result as an increase of motor activity. Many local diseases require rest to effect a cure; *e.g.*, inflammation of the peritoneum, bladder, or kidneys (surgical conditions are, of course, not included). Again, in all acute febrile diseases, in prolonged weakening conditions,

and in painful affections, rest in bed is sought, inasmuch as both strength and the desire to move grow less, and instinctively there arises a desire to curtail the expenditure of energy as the appetite diminishes. In many of the chronic diseases, such as tuberculosis, inanition, and anæmia from whatever cause, but particularly in chlorosis, rest in the fresh air (in bed or on a couch) is indicated, continuous at first, but later at intervals; and this forms a most important part of the plan of treatment. At the same time the body weight may if desired be increased by forced feeding.

Another indication for bodily rest, which should by the way always be accompanied by mental rest, is a weak heart action, whether this be due to disturbances of compensation through cardiac failure, to paralysis after scarlet fever or diphtheria, to the weakened conditions secondary to influenza, or to other causes, and this safeguard should be continued so long as exertion causes the heart to beat much more rapidly or irregularly. A final indication for prolonged rest is to be found in those diseases of the nervous system which are accompanied by mental or physical unrest (excitement, fear, marked tics, and chorea). Every rest cure must be terminated at the proper time, lest the patient become debilitated.

Even during the course of the treatment, the muscular tone may be somewhat stimulated by means of dry rubs, sponges, alcohol rubs, or light stroking massage, which must be very carefully performed in cardiac disease. To this may be added somewhat later muscular exercise in bed. The muscle exercises may be either active or passive. They must encourage respiration by being performed rhythmically, and synchronously with deep inspiration and expiration.

Massage is next to be tried with the passive muscle exercises. Strong kneading of the muscles is, as a general thing, to be avoided; it must not excite the patient or be followed by sleeplessness. General massage is next employed for a time, but its use, at the beginning, must be restricted to the extremities. General massage is also useful in the muscular weakness of flabby children and in the muscular relaxation of rickets. Its sphere of usefulness is, upon the whole, not a large one, since active exercise in the open air, under favorable conditions, is very much better, as we will show later. When this cannot be practiced, however, as in the muscular atrophies following diseases of the nervous system, massage must take its place. The massage of certain groups of muscles is indicated in orthopædic cases and in the treatment of chronic constipation. Abdominal massage, which may be practiced in various ways, but always methodically (either by the physician or by the nurse), must of course never be used in acute inflammatory conditions of the abdomen. Older children may be able to massage themselves by using an iron ball with a knitted or leather cover and weighing from

2 to 3 kg. (3 to 5 lb.). The patient lies upon the back, with the knees drawn up, and the ball is rolled around the umbilicus in the direction in which the hands of a clock move. This should be done in the morning before food has been taken, and be continued for at least ten minutes.

Electrical treatment is also to be considered. This is of very little use in childhood, excepting as it acts through suggestion, and in paralysis of muscles.

Passive movements particularly are to be practiced in such a way that they will be dichronous, and be accompanied by deep inspiration and expiration. For example, a position is taken behind the patient, who is seated on a chair, or if bedridden upon the edge of the bed; the elbows are grasped and the arms are raised from the side, or rolled in the shoulder joint, or carried horizontally forward and then backward, without, however, allowing the patient to participate in the effort. The transition to active movements is made through the use of resisted movements, in which the attempts by the patient at flexion and extension of the extremities and body are resisted by a more or less powerful pressure in the opposite direction by the hand of the operator. A skilful graduation of the pressure can call forth any degree of muscular contraction, and by gradually increasing the pressure the functional ability of the muscles is progressively improved. Passive and resisted movements may also be carried out by means of special apparatus. By this method it is possible to graduate minutely the exercise for each group of muscles. The active exercises are the more natural, however.

During early childhood, from about the second to the sixth year, there exists an innate tendency to exceedingly active exercise, which even in future years, at least in males, manifests itself in a strong desire for suitable exercise within certain limits. Different organic diseases may interfere with exercise, especially when they are accompanied by dyspnoea; nervous diseases may also act similarly. Of the latter, neurasthenia, with its hypersensitiveness to bodily sensations, takes the lead from a therapeutic standpoint; but, as will be shown below, the combined physical method of treatment may readily overcome the obstacles interposed by mental influences.

As early as the fourth year, but principally during the school years physical training is added to the natural exercise. This has a most remarkable influence upon the development of the powers of observation, the will, and the muscular control, and improves the capacity and strength of the muscles, particularly those of respiration. As we will not here consider orthopædic exercises, or exercises for simple hygienic purposes, we need hardly take into consideration exercise by means of apparatus, but may simply direct attention to those which can be performed without any apparatus excepting perhaps the horizontal bar. These are to be performed in the fresh air, in as light clothing as possi-

ble, and may even be carried out while taking an air bath (see below). Marked acceleration of the pulse and overheating must not be permitted to occur, where exercise is ordered with a purpose. For school children, swimming in summer and skating in winter are also to be encouraged; and in later youth various sports, such as tennis and bicycling. The latter is to be used moderately by weakly or nervous subjects, care being taken that they do not overexert themselves; in other words, they should not be permitted to take long journeys without a predetermined limit, and then only while maintaining a correct position upon a saddle properly shaped and adjusted.

Combined Physical Therapy.—We have now reached the consideration of the method of treatment by combined physical therapy. In an emergency we restrict ourselves to only one of the many natural and easily accessible healing agents. For instance, association with other children, or the stimulation of natural surroundings, will often be the means of inducing a child to enter into the spirit of exercise, so that it will be engaged in sufficiently, and with especial pleasure. At the same time, the association with individuals of the same age is of great value, excepting during the first two or three years of life, as it tends to develop and educate the child's mind, and neurasthenic disorders, for instance, are forced into the background. The freedom of the exercise amid natural surroundings permits these powerful influences for health to become efficient. To these influences we shall now for a short time turn our attention.

It is impossible here to bring out in detail the value of the individual points of climatotherapy; the cleanliness and the humidity of the atmosphere, its temperature, and motion; the sun, the terrestrial radiation, the barometric pressure, etc. The subject will be considered from an essentially practical point of view.

It is not always borne in mind that the greatest harm which modern culture has brought about is the restriction of life in the open, amid the surroundings of nature, with the evil results directly due to this restriction. Rickets and tuberculosis in all their forms are in great part caused by this. From earliest youth air, light, warmth, and dryness are required by the human body. Since little children cannot wander far from the homes which protect them against the inclemency of the weather, they must, if they are doomed to grow up among the stone piles of a large city, search for dust-free playgrounds. Older children can and should take plenty of exercise, and for this purpose the open spaces in the outlying districts of the city should be made use of for play and exercise. The summer camps answer this purpose for school children during their summer vacations.

When travelling in the interest of health, the drawbacks of the trip must be borne in mind (dust, trains, irregular and improper nourish-

ment, the jarring, and the excitement incidental to a journey), and it is necessary to decide whether the existing disease may not become indirectly aggravated, or whether the ultimate results, particularly in regard to a cure, may not be more readily attained in suitable home surroundings, under proper conditions. Little children should not travel far, or often; older children only under proper guidance and without being overtaxed. Travelling is to be forbidden in acute diseases. In the same way, subacute diseases of the respiratory tract, of the intestines, of the sensory organs; even many chronic diseases (*e.g.*, chronic enteritis) may often be cared for to greater advantage at home, owing to better medical and dietetic treatment. While travel under the restraint and care of members of the family is very desirable as a rule, in cases of hysteria or degenerate conditions, especially the neurasthenia and scrofula which so often exist together in the same individual, separation from the family and admission to an institution are followed by much more favorable results. In this way, too, the cure can often be carried on for a longer time. In fact, four to five weeks usually serve only to refresh weakly children, not to cure sick ones. In chronic conditions, it is necessary to extend the treatment (Kur) over many months; or, if this is not possible, courses of treatment for periods of from five to eight weeks should be repeated yearly.

A prescribed course of treatment in a health resort must not be permitted to endanger the health of the child by causing intestinal catarrh, typhoid fever, or children's diseases generally (especially whooping-cough). For this reason, in choosing the health resort, it is necessary to take into consideration the milk and water supply, the condition of the dwellings and baths, and the possibility of obtaining satisfactory medical attention. In a precipitous country a young child may easily stumble, and poor roads whose surface does not drain well owing to the impervious nature of the ground, may interfere with exercise in the open. The discrimination between watering places and climatic resorts (mountains and seashore) is only theoretical, since the prescription of baths and of drinking spring waters may be combined with climatic and other hygienic and dietetic factors, with the happiest results. In the choice of a proper climate, it may be stated that for children during the first two or three years of life, extremes are to be avoided, and even with older children residence in such a place may be recommended only for trial. Finally, the experience obtained in previous cases must not be forgotten and must be taken into consideration in making selection of a resort. The limits given for particular ages may be extended in certain diseases; *e.g.*, the true anæmias (we do not mean the regional anæmia of skin and mucous membranes) require a place where the climate is warm, without marked variations in the temperature, and without the temptation to long walks. Altitudes of

900 to 1200 feet for children during the early years of life, and of about 3000 feet for older children, should not be exceeded. Living at such a resort may be combined with baths (of carbonate of iron or other mineral waters, or of carbonic acid brine).

Anæmic patients as a rule bear the rough sea well (*e.g.*, the North Sea, or, better still, the Baltic, to which they may be sent if one does not care for the steel or salt baths situated in a warm, dry climate). The Baltic Sea is the Eldorado of little children, especially the rachitic and scrofulous. The sea climate here is of the mildest, with only light winds, which are an advantage; the atmosphere is pure, the sunlight is strong, and there is a beach of fine warm sand upon which to lie or play; and even if the sea is not used for bathing by little children, they may nevertheless paddle barefooted in the water once in a while. Some of the Baltic Sea resorts also have brine baths, and woods in which to walk. The advantages which the Baltic Sea presents in summer, are also to be found in the spring and fall on the shores of the Mediterranean. Among the Baltic resorts possessing good beaches may be mentioned Heringsdorf, Bansin, Ahlbeck, Swinemünde, Dievenow, Kolberg; most of them also have brine baths, and woods. On the Mediterranean Sea, may be mentioned the Riviera (particularly Mentone, Bordighiera, and Rapallo); and on the Adriatic Sea, Abbazia and Grado.

Scrofulous children of a more advanced age should be sent to the North Sea, where the high winds and sea baths, combined with other factors, act favorably. Here are to be mentioned Norderney, Westerland on Sylt, and Wyk on Föhr; and among the expensive watering places of Holland and Belgium, Zandvoort, Scheveningen, Blankenberghe, and the world renowned Ostend.

Sea baths must not be taken so frequently, or continued for so long a time as to cause shivering and discomfort, which are followed later by nervous excitement, sleeplessness, headache, loss of appetite, etc. They are to be begun only after the child has become acclimated, the duration and frequency of the baths to depend upon the weather and upon the individual conditions.

It has already been stated that scrofula requires repeated courses of treatment, or one course prolonged for months. This deserves repetition. The North Sea is also suitable in winter for prolonged treatments. But, as the Baltic Sea may be too great an excitant for very young nervous children (causing, for example, the appearance of enuresis and polyuria, and night terrors), so may the North Sea act upon older children. This should be guarded against, since it may cause failure of the whole course of treatment, even leaving out of consideration the possibility of asthma, etc.

Nervous children do better among the hills, rich in woodland, where the atmosphere is stimulating (*e.g.*, the Black Forest, Schierke in

the Harz Mountains, Oberhof in Thuringia, Berchtesgaden in Upper Bavaria), or even in the mountains (Switzerland and the Tyrol), where they should be kept away from the hotel life as much as possible, permitted to take moderate walking trips, and, for the rest, be kept as quiet as possible. Of course, remedial measures (indifferent or carbonic acid baths, or baths in inland waters, streams, etc.) may be employed here in the same manner in which simple hydropathic procedures are carried out.

For the many sick or sickly children who ought not to be exposed to stronger influences, there still remain those resorts which are at sea level or only a few hundred feet above it (*e.g.*, Mecklenburg, the Saxon Switzerland, the Harz Mountains, and Thuringia). Owing to the penetration of the sun's rays and to the shade and fragrance of the woods, it is possible in such a place to often supplement the action of the mineral springs, the indications for which latter have been given above,—rickets, scrofula, cardiac and renal diseases, rheumatism in its various forms, etc.

A simple sojourn in the country may prove beneficial and produce excellent results, even when nature does not appear to offer any particular local advantages for the systematic use of the climatic influences. The air bath, for instance, produces a mild but beneficial effect upon the skin; this may be used to advantage in the mountains and at the seashore; warmth, light, and water may all be utilized. The stimulating effect of association with other children, too, is brought into play at health resorts, so that in many cases the result of the treatment is very good.

While the well-to-do travel with their children when a cure is ordered, the children of the poor must be grouped into camps, during their vacations, when the course of treatment is only a short one (about four weeks). But, beside the vacation camps, there are sanatoria which are open during the winter as well as the summer (like the Sea Hospital at Norderney), where the course of treatment is usually longer (six to eight weeks, but rarely more).

In Germany children's sanatoria are to be found in the following places: *Brine and carbonic acid brine baths* at Alstaden, Arteren, Bernburg, Dürkheim, Dürrheim, Elmen, Frankenhausen, Goczalkowitz, Halle, Harzburg, Hohensalza, Jagstfeld, Kissingen, Königsborn, Königsdorf, Kösen, Kolberg, Kreuznach, Lüneburg, Nauheim, Lower Neukirch, Oldesloe, Orb, Rappenu, Rothenfelde, Salzdetturth, Salzußen, Sassendorf, Schwäbisch Hall, Sooden, Sülze, Sulza. *Seabaths*: Berg-Dievenow, Duhnen, Gross-Müritz, Heringsdorf, Horst, Kolberg, Norderney, Olgaheim am kleiner Tinnerndorfer Strand, Rügenwaldermünde, Travemünde. Others are to be found in Brandenburg, in Lychen, and in Dyrotz. The management of these institutions is not uniformly good, varying from those kept by laymen in a most unhygienic manner, to

those which are conducted along ideal lines under the supervision of a resident physician. The number of institutions in which children of the well-to-do may be placed is extremely small, yet it is among such that a separation from the parents is often most desirable.

The success of a course of treatment may evince itself in either the local or the general condition: the symptoms of scrofula (*e.g.*, eye disease and eczema, inflammatory exudation, and rheumatic effusions) disappear; tuberculous or scrofulous glands diminish in size; and in the severe scrofulous conditions (*e.g.*, of bones and joints) a relative cure may be attained if the necessary medical supervision has accompanied the course of treatment, which must be continued for months, or even for years. Conditions of exhaustion, anæmia, and chlorosis are often improved in a surprising manner by means of a suitable course of treatment. To have been successful, however, every such course of treatment must have raised the spirits and the general tone, produced greater elasticity of carriage and motion, and have increased the powers of endurance.

As a rule, too much importance is laid upon a simple increase in weight, although this usually occurs, and may be taken as an index of the success of the treatment. It must not, however, be taken by itself in judging of results. The amount of increase in weight depends upon the state of nutrition at the beginning of the course of treatment, upon the nature of the disease, and upon the age and sex of the patient, as well as upon the time of the year. Younger children increase less than older ones; girls more than boys; and girls at the time of puberty, when natural development is at its height, show the greatest gain of all.

Pharmacotherapy.—A difficult problem for the physician to overcome is the necessity of prescribing drugs. This tendency should be counteracted by the enlightenment of the public, and the development of the physician upon the subject of general therapeutics. It is in pediatrics especially that the physical and dietetic measures are often more important than the chemical.

The loading of remedies in a single prescription, often caused by adherence to an inherited formula, not only makes a critical judgment impossible, but also increases the cost of the medicine, thereby preventing the poor from treating their children. Moreover, this is frequently absolutely incorrect, since important constituents may remain undissolved (*e.g.*, alkaloids in alkaline solutions), or new combinations may be formed (alkalies forming salts with acids, or reductions or oxidizations may occur). By the addition of correctives, unintentional collateral effects may occur, diarrhoea may result in young children, for instance, if fermentation has begun in the syrup which is used.

Again, the use of an ill-tasting medicine (the physician should have tasted, at least once, every medicine he may prescribe) may at

the very outset of the treatment arouse antagonism in the child, which is all the more undesirable in that it might have been avoided. The medicine also may interfere with the taking of nourishment. The efforts of pharmacists to produce drugs in compressed form of exact dosage, or in tasteless chemical combinations, are of especial value for children. The tablets should be dissolved before administering them, inasmuch as they cannot be swallowed by children any more than can pills or capsules. In prescribing the tasteless combinations, it is necessary to take the bulk of the dose into consideration. The prescribing of decoctions, emulsions, and even infusions may be avoided, or at least minimized, by giving preference to solutions, extracts (fluid), and powders.

The time for the administration of medicines should be regulated by the contemplated action of the drugs. If, for instance, it is desired to treat a mild catarrh of the mucous membranes, it is necessary to bring the medicament into contact with them frequently (*e.g.*, about every two hours). If the body is to remain under the constant influence of a drug, the frequency of the dose must be governed by the rapidity with which it is excreted; iodides, for instance, should not be administered less frequently than three times daily, and phosphorus not more than once. Some drugs do not produce their full effect until a certain quantity has been taken, and it therefore becomes necessary to administer them at short intervals until the effect becomes perceptible. Digitalis and veratrum viride are drugs of this character. Occasionally, it is necessary to give only one large dose to obtain an effect, as when it is desired to produce sleep or vomiting.

It is necessary to consider the relation of medicines to the meals. Those which are intended to stimulate the appetite should be given before meals; those which may easily irritate the mucous membranes of the stomach (*e.g.*, iron and arsenic), toward the end of the meal; and those which should enter the intestines unaltered (*e.g.*, anthelmintics, or drugs intended to combat an intestinal catarrh), should be given when the stomach is empty.

Ill-tasting medicine when mixed up in food and administered to little children is a source of future trouble since the child will distrust foods offered, imposing additional difficulties in feeding. It is better to give the fluid, or a powder floated upon water, by force if necessary. The spoon is not to be placed upon the lips, but carried far into the mouth; a certain amount of pressure is to be exerted on the back of the tongue, and the medicine is to be introduced slowly, drop by drop, until a swallow has been taken, this being repeated until the spoon is emptied. In older children it is possible to administer the whole dose at once by pinching the nostrils until the mouth is opened, pouring in the entire dose, and not withdrawing the spoon until after the medicine has been swallowed.

The regulation of the dose of medicine to be employed is not difficult, when rapidly repeated small doses are to be employed for an easily recognized effect. If, for instance, it becomes necessary to coat the gastric mucous membrane with bismuth, or the intestinal mucous membrane with an almost insoluble preparation of tannic acid, large doses of these harmless remedies may be administered without interfering with the desired action. One may give as much as one-half to 1 Gm. (seven and one-half to fifteen gr.) of a tannic acid preparation every hour, for 4 doses. When, however, as in the various digitalis preparations, the action is cumulative and continues after the effect of the drug can be recognized, it is necessary to use small doses carefully, so that the amount of the drug ingested will not produce more than the desired effect. It is necessary, however, not only to administer drugs in such a manner as to obtain the maximum chemical and therapeutic results, but also, by proper dosing, to limit the collateral effects. For instance, ipecac may produce intestinal irritation before its action as an expectorant is thoroughly manifested. The collateral action, however, may usually be prevented by observing proper precautions at the right time; *e.g.*, in using mercury, the mouth should be kept clean and the action of the bowels be regulated, and in the use of iodides or bromides, table salt in the food should be restricted to a minimum. When idiosyncrasies exist, as against antipyrin or opium, it is necessary to desist from its use in even the smallest doses. Only in rare instances is a case met with in which a patient is oversensitive to an entire group of remedies.

Certain drugs may have opposite actions even when used within the normal limits of clinical therapeutics, as when calomel, castor oil, rhubarb, or Glauber's salt used in small doses produce constipation, and in larger doses act as cathartics. Glauber's salt is present (only 0.239 per cent.) in Karlbath Mühlbrunnen water, which constipates when administered to children in quantities of 20 to 100 Gm. (five to twenty-five drams) for gastric and intestinal catarrh, whereas only one-half to one dram is required for a laxative effect.

When adult doses are taken as a standard, it is not sufficient to use the age or weight in arithmetical comparison. Still, the latter gives a very important clue for graduating the doses of various remedies (*e.g.*, the alkaloids and nerve poisons of all kinds); while such remedies as astringents, expectorants, cathartics, etc., whose principal action is local upon the internal mucous membranes, must be used in relatively larger doses for children. In childhood, especially during the first and second years, the size of the dose of medicine must be carefully considered. The dose is to be gradually increased during the following years (without assigning any particular dose to each year, however) until, between the ages of ten and fourteen years, the size of the dose is about one-half that of the adult. The physical development, however, must

in a measure be taken into consideration. For the first six or eight weeks of life, all internal medication should be avoided, as a rule, excepting in such conditions as congenital syphilis, or spasms. During the remainder of the first year, great care is necessary in the administration of drugs, especially opiates (one minim of the tincture of opium may be fatal); whereas sedatives are well borne,—*e.g.*, chloral hydrate (up to 0.5 Gm. (seven and one-half gr.) once daily internally or by rectum) and bromides (up to one Gm. (fifteen gr.) per day). An uncertainty in the amount administered arises through the poor method of ordering a “teaspoonful,” “as much as may be held on the point of a penknife,” etc. Even though the capacity of a teaspoon is in general between 3 and 5 Gm. (forty-five and seventy-five gr.), a dessertspoon between 8 and 10 Gm. (two and two and a half drams), a soup spoon between 12 and 15 Gm. (three and four drams), the point of a penknife between 0.5 and 1 Gm. (seven and a half and fifteen gr.), the specific gravity of the remedy and the degree of care with which it is dispensed may increase these variations considerably. Because of the simplicity with which they may be figured, it is customary to consider a teaspoonful as 5 Gm. (75 gr.), a dessertspoonful as 10 Gm. (two and a half drams), a tablespoonful as 15 Gm. (one-half ounce), and in so doing the dose actually given is somewhat less than the calculated dose. The measuring of fluid medicines in medicine glasses, and the dispensing of dry drugs in exactly weighed powders is to be preferred for certain drugs. If medicines are ordered in drop doses, one should figure twenty drops to one gram (which is sometimes very inexact).

For a long time pharmaco-therapeutic research devoted itself to its task in a very unsatisfactory manner, since drug action was studied upon the healthy human or animal organism, whereas the goal can be approached only from the new direction taken by experimental therapy, that of influencing experimentally the cause of the disease, or the disease process itself. So the laboratories unite in their efforts to add, in the form of organ and serum therapy, new specifics to the few (*e.g.*, mercury, iodine, and quinine) which observation at the bedside gave to us long ago. Of the innumerable drugs which act only upon certain organs, or systems of organs, many are of real value, while others owe their vogue only to tradition. That the latter, because of their empirical basis, should be cast aside without further ado is hardly to be advised, when one considers, for instance, the increase in weight which follows the improvement of appetite (all other conditions remaining unaltered) produced by the administration of a bitter remedy (*tinctura cinchonæ composita*) in spite of the fact that up to the present time experimentation can offer no explanation of its action. And finally, it cannot be wondered at, that the action of many remedies—of water cures, for instance—is not entirely clear, inasmuch as under certain circumstances

the functions may be restored to normal through influences which are almost imperceptible and outwardly are scarcely recognizable. If we have previously, among the physical methods, learned to appreciate the action of the bath, we have no right to deny an increase of its activity through the addition of chamomile tea, because the manner of this action is unknown to us excepting through the sense of smell. It is impossible to know the details of the action of a large number of remedies, used for identical purposes, and if the selection is merely a matter of fashion, it is always well to take into consideration the degree of toxicity and the price.

MORTALITY AND MORBIDITY IN INFANCY

BY

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MORTALITY varies greatly according to age. By grouping the deaths in a community so as to ascertain the rate per 1000 for each age, we may construct a table of mortality giving a clear idea of respective death rates.

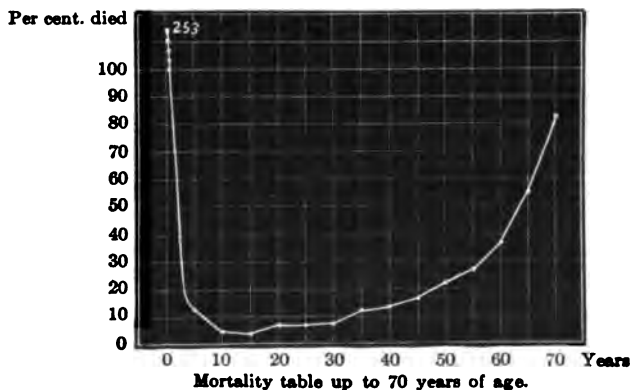
According to the tables, the mortality is very high in the first year, is at its minimum from the tenth to the fifteenth year, and increases in

TABLE 1.

Age.	Deaths.	Age.	Deaths.
0 year (Born living)	253	35 years	11
1 year	65	40 years	14
2 years	33	45 years	17
3 years	23	50 years	21
4 years	17	55 years	28
5 years	13	60 years	38
10 years	6	65 years	56
15 years	4	70 years	81
20 years	8	80 years	174
25 years	8	90 years	319
30 years	9	100 years	519

Mortality table, showing number of deaths per year in every 1000 persons.

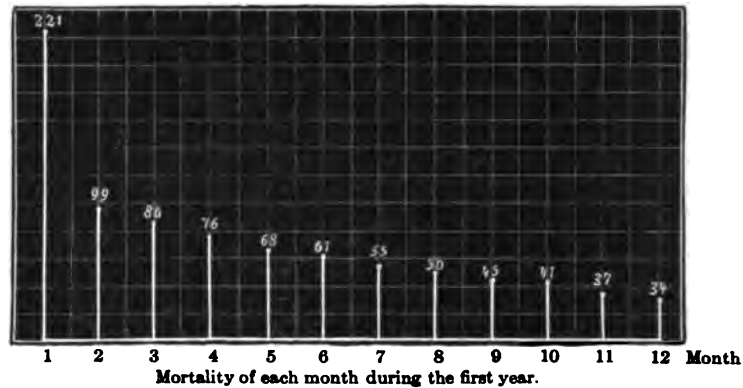
FIG. 17.



the succeeding decades, at first slowly, and later more rapidly. This may easily be seen from figures taken from the German Table of Mortality of 1871-81 (Table 1) and from the curve (Fig. 17).

The mortality *during the first year* is by no means uniform. A calculation in the Berlin Statistics of 1893-1897 (Westergaard) gave the figures shown in the diagram (Fig. 18). The death rate, high in the first month, falls rapidly at first, and then falls more and more slowly. Of 100,000 children of the respective ages the average daily mortality was:

FIG. 18.



A more minute analysis of the mortality *during the first month*, distinguishing boys and girls and between legitimate and illegitimate children, yields the figures in Table 2. From these it may be seen that the mortality of boys is greater than that of girls and that the mortality of illegitimate children is almost double that of legitimate.

TABLE 2.

Time of death.	Legitimate.		Illegitimate.		Unclassified.
	Boys.	Girls.	Boys.	Girls.	Both sexes.
Still-born	326	268	536	496	330
Found dead	...	...	43	43	6
1st day	121	92	244	207	154
2d day	56	40	111	90	56
3d day	33	22	66	46	32
4th day	19	15	38	35	20
5th day	15	10	32	23	15
6th day	14	12	32	23	15
7th day	16	12	30	28	16
1st week (total)	274	203	596	496	284
8th to 15th day	113	88	236	220	119
16th day to end of month	187	157	516	432	217
1st month (total)	574	448	1348	1147	620
Still-born and total deaths	900	716	1884	1645	960

Still-born and other deaths during the first month among 10,000 births.

The above figures, showing the high mortality of children during the first year, have been taken from German statistics, but the same phenomena appear everywhere, though in varying degrees. We adduce as proof of this a table calculated by F. Prinzing, in which the mortality of children in the different European countries is compared (percentage

of children who die in their first year, omitting the still-born). Beside this we tabulate the number born per 1000 inhabitants, the number of these that survive their first year, and the number that die after their first year and before the expiration of their fifth (Table 3). It may easily be seen from this table that the effect of infant mortality on population depends partly on the ratio of births. In France, for example, in spite of the low death rate among infants, the number of survivors is not so great as in Germany, because the number of births is smaller. The table also shows that it is an error to believe that a high death rate among infants is advantageous because it eliminates the weak. Countries with a low mortality in the first year usually have also a low mortality among children between one and six years old.

TABLE 3.

Country.	Birth rate of living children per 1000 inhabitants 1881-1890.	Number surviving the first year.	Mortality of children between the ages of one and five years.	Mortality of children, 1884-1893.
Italy	37.9	30.5	18.6	19.04
France	24.1	20.1	9.8	16.71
Switzerland	28.2	23.5	7.0	16.37
Belgium	30.0	25.2	9.7	16.29
Holland	34.2	28.1	10.3	17.50
Prussia	37.1	29.4	13.1	20.79
Bavaria	35.7	25.6	11.9	27.90
Saxony	41.9	30.1	11.5	28.28
Württemberg	35.8	26.2	9.8	26.14
Austria	38.1	28.6	16.6	24.91
England and Wales	32.6	28.0	9.8	14.64
Scotland	32.4	28.5	10.0	12.23
Ireland	28.4	21.2	7.6	9.63
Sweden	29.0	25.8	8.3	10.71
Norway	31.8	28.7	9.3	9.51
Denmark	32.1	27.8	6.9	13.42
Finland	34.8	29.5	12.9	14.91
Spain (1878-1882)	36.3	29.3	26.0	19.17
European Russia	49.3	46.4	...	26.79

We see by Prinzing's tables that during the last century there has been a considerable decrease in infant mortality in Switzerland, Holland, Italy, Finland, Sweden, and Norway. In the other European countries the fluctuations are slight. An increase in the middle of the nineteenth century was followed by a decrease, which in Austria was permanent. In Belgium, Great Britain, and Ireland an increase was visible in 1886-1896. France, Denmark, and Russia have, in the last two decades of the century, shown a uniformly high ratio.

In the different sections of Germany, infant mortality varied greatly. From 1871 to 1875 there was a marked decrease in the South, accompanied by a far greater stability in the North. It is significant that in Prussia a decline in mortality was shown for all except infants. This must be attributed to the improvement in hygienic methods and in the standard of living among the poorer classes, effected by social legislation. This decline is made apparent in a table by Kruse, which is here given in part. In the last quarter of a century the mortality of

infants has remained almost constant, while the mortality of older persons has decreased nearly one-half.

Under such circumstances, infant mortality demands special attention. We must first of all determine what diseases occasion the heavy mortality of the first year. In the following diagram (Fig. 19) we may find the most important diseases, according to the German statistics for

TABLE 4.

Quadriennial period.	Average of the year.						Average of all ages.
	0 to 1	1 to 2	2 to 3	3 to 5	5 to 10	10 to 15	
1875-1879	266	71.5	37.6	22.7	9.4	3.9	27.4
1880-1884	271	71.9	36.6	21.6	9.5	4.0	26.8
1885-1889	267	69.6	34.6	20.2	8.3	3.7	25.7
1890-1894	266	63.7	29.9	17.8	7.3	3.2	24.6
1895-1899	267	54.9	21.8	12.0	5.0	2.7	22.6

Showing number of deaths per 1000 persons in Prussia up to fifteen years of age.

1900, and will see that, for every 1000 births, 225 deaths occurred in the first year. Of these 225 deaths, 84, or more than one-third, were caused by stomach or bowel troubles. Diphtheria and croup, whooping-cough, measles, and scarlet fever, together, account for only one-twentieth of the entire mortality; and tuberculosis, inflammation of the lungs, and other inflammatory ailments of the respiratory organs, for about one-

FIG. 19.

Congenital weakness and debility. 32	Gastro-intestinal diseases. 84	Diphtheria, 2. Pertussis, 6.4. Measles, Scarlet Fever, 3-11.4.	Tuberculosis, 2.4. Pneumonia, 8.6. Other inflammatory respiratory diseases 10.5-21.5.	Other known diseases. 68	Unknown diseases 8.0.

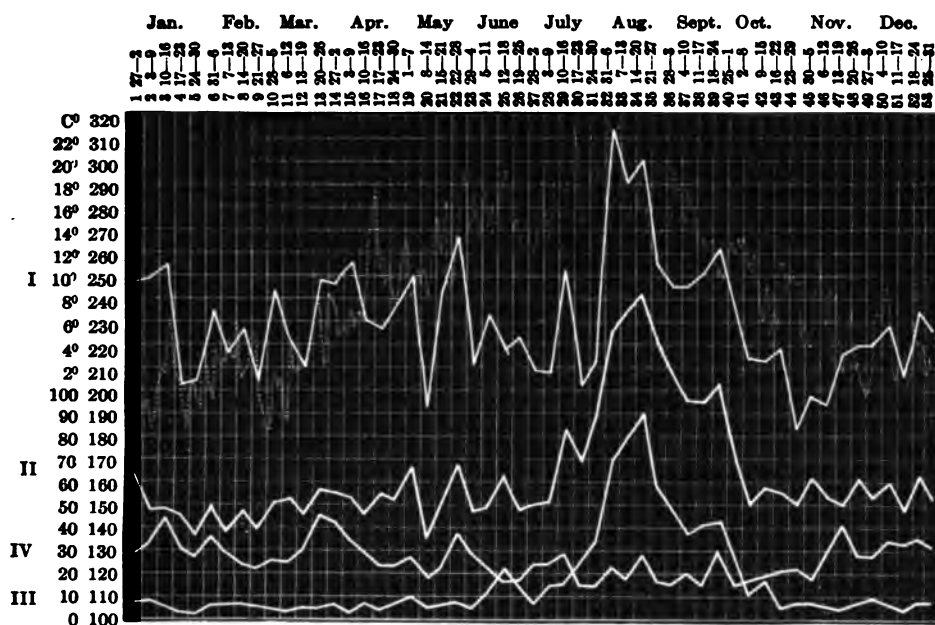
Causes of death of 1000 children dying in the first year of life.

tenth. This prominence of the stomach and bowel troubles may be clearly exhibited in another way. Fig. 20 shows, according to the Hamburg statistics of 1904, (I) the actual number of weekly deaths, (II) the deaths of children in their first year, (III) the deaths by diarrhœa and cholera, and (IV) the deaths from acute ailments of the respiratory organs. From this diagram it is evident that the general mortality for the year is determined by the infant mortality, and this, in turn, by the number of deaths arising from diseases of the digestive organs. The inverse

mortality curve of the more or less numerous diseases of the respiratory organs (curve IV) cannot materially affect the general result.

It is therefore entirely natural that the statistics of infant mortality are concerned more particularly with the *diseases of the stomach and bowels*. We must not fail to consider the objection often raised to statistical tables of deaths from diseases of the alimentary canal, that the data are untrustworthy. Apart from the fact that many infants die without medical attendance, and so without a professional determination of the cause, even the calling in of a physician affords, in many cases, no guarantee that, in case of death, a correct diagnosis will be given.

FIG. 20.



Mortality curve of the 52 weeks in the year. Dotted lines show the daily temperature of the air.

The importance of such defects in the data is clearly shown by a compilation by Würzburger, published by Schlossmann. According to this, out of 8600 children under one year old, who died during the years 1891-1896, the cause of death was certified by a physician in only 4245 cases, or 49½ per cent. According to von Mayr, it may be seen from the general report on sanitary administration in Bavaria that, in the very places where the death rate among children is the highest the percentage of medical attendance is the least. In the year 1893, for example, in 10 districts with an infant mortality of from 40 to 60 per cent. only from one to eleven per cent. of the children received medical attendance. In the district with the highest mortality, 46½ per cent., only one and one-tenth per cent. of the cases were attended by physicians. The value of the data is materially impaired by this fact.

A mortality curve essentially the same as that of the Hamburg statistics may be obtained wherever artificial nourishment of infants predominates, and even where many are nursed at the breast. This holds good especially when breast-fed babies are given other forms of nourishment.

Researches into the *influence of nutrition* on infant mortality are very numerous. Boeckh has analyzed the Berlin statistics along these lines with the greatest care. Not only has he inquired into the nutrition of every deceased infant, but he has also caused the various methods of nutrition of living infants to be ascertained at each census. The survivors for each month, as given in the Table of Mortality, have been classified according to the data of nutrition in the census. In the same way the deaths at each age have been classified according to the statistics of mortality. Westergaard considers that only still-born infants and those dying on the day of their birth should be disregarded. For the years 1895-1896, so far as the two chief means of nutrition are concerned (milk from the breast and milk from animals), the following figures are obtained. Of 10,000 children there died in each month:

TABLE 5.

Age.	Breast-milk.	Cow's milk.	Age.	Breast-milk.	Cow's milk.
0 month	201	1120	6 months	26	277
1 month	74	588	7 months	24	241
2 months	46	497	8 months	20	213
3 months	37	465	9 months	30	191
4 months	26	370	10 months	31	168
5 months	26	311	11 months	39	147

The mortality of children nursing at the breast is thus considerably lower than is the mortality of those fed artificially. The following table compiled by Westergaard is also instructive. It shows

TABLE 6.

Age.	Breast-milk.	Cow's milk.	Milk substitutes, whether or not combined with breast-milk or cow's milk.
0 to 1 month	10	32	38
1 to 2 months	23	52	62
2 to 3 months	29	52	56
3 to 6 months	26	50	59
6 to 9 months	18	42	56
9 to 12 months	13	30	39
0 to 1 year	17	43	53

Showing form of nourishment per 100 deaths due to intestinal diseases.

the predominant influence of the mortality from digestive diseases where children are fed on animal milk, and particularly when they are fed on substitutes, whether these are used in combination with milk or not.

Among the further details of Boeckh's researches, the determination of the influence of the method of nutrition in the case of legitimate and illegitimate children deserves especial attention, as will be seen by referring to Table 7.

The influence of *illegitimate birth* upon the well-being of infants and children has been made the subject of numerous statistical inquiries, and always with the result that the illegitimate are found to die in greater numbers. In extending such statistics over a protracted period,

TABLE 7.

Age.	Legitimate.		Illegitimate.	
	Breast-milk.	Cow's milk.	Breast-milk.	Cow's milk.
0 month	196	1028	267	1252
1 month	76	580	143	915
2 months	64	544	63	887
3 months	58	478	75	801
4 months	49	441	46	720
5 months	44	424	31	525
6 months	42	444	80	417
7 months	47	325	26	389
8 months	50	282	38	363
9 months	47	259	45	260
10 months	59	218	81	276

we must bear in mind that a considerable part of the children born out of wedlock are eventually legitimized. According to Würzburger, during the years from 1894 to 1898 barely a third of all illegitimates in Dresden attained their sixth year without being legitimized by the marriage of their parents. Boeckh, in order to eliminate this source of error, carefully scrutinized the mortality records at Berlin, and obtained the following results for the year 1885:

TABLE 8.

Age.	Legitimate.	Illegitimate.	Age.	Legitimate.	Illegitimate.
At birth	908	943	10 months	757	543
1 month	911	828	11 months	745	530
2 months	889	767	12 months	735	515
3 months	868	716	1½ years	709	488
4 months	847	676	1½ years	691	471
5 months	830	638	1½ years	679	455
6 months	813	613	2 years	669	446
7 months	796	590	3 years	642	425
8 months	782	572	4 years	624	412
9 months	769	557	5 years	612	405

Showing number of legitimate and illegitimate children surviving per 1000 births, up to five years of age.

The mortality of illegitimates is at first strikingly greater than is that of legitimates, but it decreases constantly until illegitimates from 4 to 5 years old show a power of resistance nearly equal to that of legitimate children of the same age.

The figures given for *foundlings* are even more unfavorable. Their first-year mortality, particularly with artificial nutrition, amounts to 66½ per cent. These conditions, however, have greatly altered. Found-

ling asylums have in most cases materially improved, and the mortality has consequently greatly diminished.

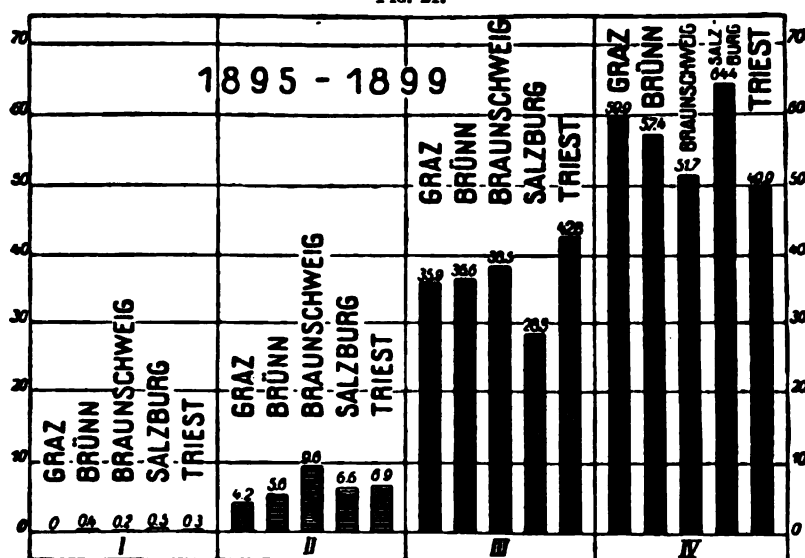
Another factor in infant mortality is the *hygienic surroundings*, as revealed by statistical research. In 1886 Meinert produced in Dresden a painstaking statistical work along these lines, devoted especially to the investigation of cholera infantum. His inquiry did not include all the deaths from diseases of the stomach and intestines, but was directed particularly to this form of cholera, with its characteristic symptoms. The value of this work arises from its careful collection of the data. Each case was elucidated by inquiry in the family and by determining the proper conditions for recovery.

Meinert has discovered, as the consistent result of all his inquiries, that the mortality from cholera infantum depends indirectly on the temperature of the air and directly on that of the dwelling. If the temperature falls below 15° C. (40° F.), in the shade, the disease seldom, or never, appears; while long periods of high temperature greatly favor its increase. Meinert's statistics show that it is not solely the excessive temperature, or the lack of cubic space per capita, in the home, that occasions the high mortality. A total temporary cessation of spontaneous ventilation in the houses, arising from their construction and occurring only under certain barometric conditions in mid-summer, constitutes the decisive factor. The number of cases decreased at once with a strong wind and increased rapidly as soon as the wind fell again. Schlossmann objected to Meinert's idea from the standpoint of the general statistics of infant mortality. Prausnitz, on the other hand, has adopted Meinert's view, in so far as it accords to the home an important part in opening the way to digestive diseases in the earliest period. In doing this he relies on the statistics of Gratz, where each single case was made the subject of a special inquiry. Statistics have still much to accomplish along these lines. The goal will be reached more quickly if the individualistic method of Meinert and Prausnitz is adopted, and an inquiry is made in the case of every infant, into all the factors that have caused its death.

Incited by earlier researches, Prausnitz has recently made protracted statistical inquiries with the aim of determining the *influence of prosperity* on the mortality of infants dying from ailments of the stomach and bowels. The deaths have therefore been divided into four categories, in accordance with a scheme proposed by Korosi,—(1) Rich, (2) Middle-class, (3) Poor, and (4) Destitute,—and the percentage of deaths from these diseases has been calculated for each category. By this means it was shown that in Gratz, for example, in the last fifth of the past century not one child died in a rich family; while in the second category about 4 per cent., in the third 36 per cent., and in the fourth 60 per cent., fell victims to diseases of the stomach and bowels.

Analogous investigations in other cities give approximately the same result, as may be seen by Fig. 21. It is a well known fact that poor people have more children than the rich, and that therefore the children of the poor die in greater numbers. A supplement to the statistics, giving the number of deaths in each category, in proportion to the number of living infants, is greatly to be desired. This is unfortunately impossible because there are no continuous records accessible which compare the number of living infants in the different cities with those belonging to the respective categories. But the researches mentioned above are by no means rendered worthless by these defects. We must, however, be careful to draw from them only the conclusions which the statistics

FIG. 21.



Influence of social condition on mortality from gastro-intestinal diseases.

logically admit; namely, that there is a close relation between poverty and the high infant mortality from diseases of the stomach and bowels. This is a most important consideration in a choice of weapons for fighting these diseases.

While, in all civilized countries, accurate tables of mortality are accessible and the accumulated data have been more or less thoroughly examined and criticized, there are no comprehensive statistics of disease. Means for ascertaining the pathological conditions of the population on a given day are lacking; so are also a continuous tabulation and utilization for statistics of all cases of sickness among the people, together with their course and outcome. The creation of such statistics has peculiar difficulties. Without the presence of a physician, a trustworthy determination of the sickness and its duration is impossible; physicians are not always called in; and, finally, the very classes which usually

call a physician have little sympathy with his wish to use their own cases for literary purposes. Hence, the only systematic records are those of infectious diseases, of which notification is required by the authorities for hygienic reasons.

In addition, there exist statistical records of individuals who have been received in hospitals or other public institutions. Finally, the data of the sick fund must be taken into account. So far as the diseases of childhood are concerned, there are no treatises covering the great mass of statistical material.

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I. MICROSCOPICAL EXAMINATION OF MILK

NORMAL milk appears under the microscope to be composed of free, structureless milk globules, which contain the fat. These circular disks become angular in cooling and present a corrugated surface, because the fat, which at an earlier stage was still in process of cooling, has grown rigid. They are now cream globules. The diameter ranges between 0.9 and 22 microns (red corpuscles of human blood 5 to 9 microns). In a microscopical view of undiluted milk, when it is rich in fat, the globules of medium size, from 2 to 5 microns, predominate. The smaller sizes become prominent only in diluted milk or in that which contains but little fat. As many as 11 millions of globules may sometimes be counted in a cubic millimetre; the average number is about 5 millions. The proportion of the different sizes is nearly uniform. In cow's milk 4 to 10 per cent. are over 4 microns, 25 to 30 per cent. are over 2 microns, 60 to 69 per cent. are under 2 microns.

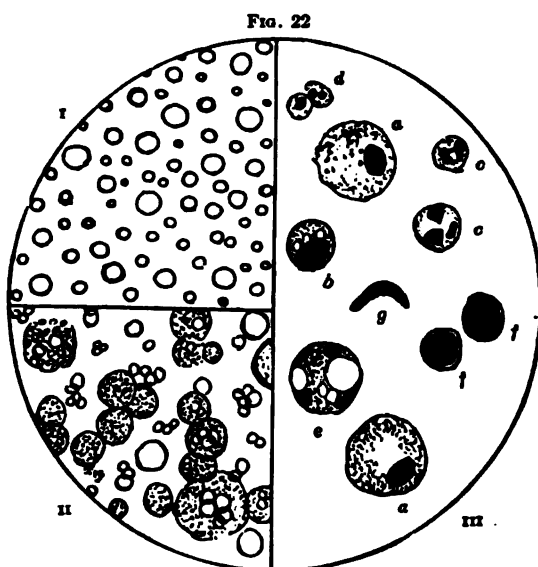
For such investigations, which have not yet been extended to human milk, the milk must be diluted a hundred-fold and one drop placed under the microscope. The size of the globules is ascertained by the micrometer.

Hooded milk globules having a fringe or surrounded by protoplasm, are frequently found, along with leucocytes, in the creamy part of human milk, and invariably so in its centrifugalized sediment, but more rarely in the milk of animals which give a copious yield. By the addition of acetic acid, or by coloring with methylene blue, a flattened nucleus is generally brought into view. We have here a trace of the characteristics of the colostrum stage, which does not, however, justify any conclusion as to the quality of the milk. A too abundant appearance of these and other constituents of colostrum, whose presence is shown also by the strength of the reactions with superoxides, peroxides, and reducing agents, indicates abnormal conditions preceding the colostrum period itself. These conditions, however, might originate in some defect of the lacteal glands or be produced later by failure to exercise the glands.

Colostrum contains in addition (1) the peculiar colostrum corpuscles, which are large, dark cells filled with fat globules of all sizes. After

their removal by ether, a protoplasmic vacuole, having a large nucleus which is faintly colored by the pigment cells, makes its appearance. (2) Mononuclear or polynuclear leucocytes, highly colored. Whether the first named, when they contain drops of fat, are to be reckoned as colostrum corpuscles is, for the present, undetermined; some at least are found which resemble colostrum corpuscles in the slight coloring of the nucleus. (3) Lymphocytes; and (4) clotted portions, which receive a faint color from the pigment cells and are turned blue and red by iodine and sulphuric acid respectively.

The microscopical investigation of human colostrum has received



Fresh Preparation.

- I. Milk globules, mostly angular as a result of cooling.
 II. Colostrum.
 III. Prepared slide of colostrum:
 a.—Colostrum bodies. b.—Mononuclear leucocyte with fat vacuoles. c.—Polynuclear leucocytes. d.—Smaller forms of the same. e.—A polynuclear leucocyte with fat vacuoles. f.—Lymphocytes. g.—Half-moon forms.
 (After Weill and Thevenet.)

some practical significance through the assertion of Weill and Thevenet that a prediction as to the subsequent yield of milk can be based upon it.

To demonstrate centrifugate 1 to 2 c.c. of colostrum, shortly after delivery. After diluting with a physiological solution of sodium chloride, remove the layer of cream, decant the milk, draw up the sediment by means of a pipette, and spread it evenly on a microscope slide. This smear is placed for 24 hours in a mixture of equal parts of alcohol and ether, stained for 15 minutes in alum hæmatoxylin and for 7 minutes in a rose-colored watery solution of eosin. If there

are more than 70 per cent. of polynuclear leucocytes the prognosis is favorable, and if more than 50 per cent. of lymphocytes it is unfavorable. In a majority of cases, however, the figures admit of no positive prediction.

The constituents of colostrum disappear in women as soon as the secretion of the milk and the suckling of the infant are well in progress, which is usually by the end of the first week. It is important to differentiate these constituents from those of inflammation of the mammary gland and duct. In this disease numerous polynuclear leucocytes and micro-organisms are present in the milk. (Testing the first drops is useless.) Fever may at first be absent.

Microscopical investigation of human milk—leaving out of the question the knowledge to be acquired of the properties of colostrum—has a practical value only in so far as a sufficient abundance of fat proves the quality of the milk. Poverty in fat, which is evidenced by the appearance of very small fat globules, cannot be inferred from microscopic examination of the first flow of milk.

In counting the fat globules, it is best to employ the stage usually used for counting blood corpuscles, taking the necessary precautions.

The microscopic examination of cow's milk sometimes reveals the presence of bacteria.

II. THE CHEMISTRY OF MILK

The fat found in milk globules consists of mixed glycerides; that is, glycerin in combination with various fatty acids; *e.g.*:

$C_4H_8O_2$	Butyric Acid
$C_{16}H_{32}O_2$	Palmitic Acid
$C_{18}H_{36}O_2$	Stearic Acid

Such a compound may be separated from butter by simple processes. The composition of the fat molecule is more complicated, since as many as fourteen fatty acids may be obtained from milk fat, and these may possibly be represented proportionately in every molecule. The only difficulty arises from the data which go to show that the smallest milk globules yield more oleic and fewer volatile acids than do the large ones. In any case, existing methods afford as yet an imperfect insight into the composition of milk fat, which, moreover, possesses a specific character. The question has hardly as yet been raised as to whether it is immaterial to the organism from what source it receives its supply of fat or whether only the quantity of the different fatty acids is important. In a treatise on milk nutrition this question would play an important part. From experiments in the fat-using industries, the cholesterin contained in milk fat seems to be an important factor in the quality of the emulsion. A small portion of fatty acids (about 0.06 Gm. per litre of milk) is present, not as a simple glyceride, but as lecithin, which is perhaps even combined with a proteid as lecith-albumin. This need not necessarily exist in the milk globules, but the larger part is supposed to pass into the cream as it rises. Besides this still disputed case, we know of three proteids found in milk, the casein, lactalbumin, and lactoglobulin, to which, apparently, there should be added the lactomucin, from which a carbohydrate is said to be split off by acids. The lactomucin appears to be present more abundantly in colostrum and in centrifugal sediment. The so-called opalisins, on the contrary, are nothing but the casein remaining in solution after precipitation.

The caseins are acid-like proteids containing phosphoric acid (nucleo-albumin or phosphoglobulin), which through rennin undergo

an unexplained change of such a nature that their combinations with lime, which do not give an alkaline reaction, are precipitated by a solution of lime salts at body temperature (rennin coagulation). They are distinguished—though only cow casein has been tested—from most proteids by the absence of a carbohydrate group. Recently, however, by treatment with ozone, there has been obtained from casein a reducing substance, forming an osazone, which may be a sort of carbohydrate.

The following table compares the proportion of the proteid-nuclei in cow casein with that of these nuclei in the globin found in the hæmoglobin of horses:

Percentage of	Casein.	Globin.
Alanin (Aminopropionic acid)	0.9	4.2
Leucine (α -Aminoisobutyric acetic acid)	10.5	29.0
α -Prolin (α -Pyrrolidincarboxylic acid)	8.1	2.3
Phenylalanin	8.2	4.2
Glutamic acid (α -Aminoglutaric acid)	10.7	1.7
Asparagin (Aminosuccinic acid)	1.2	4.4
Cystin (α -Amino- β -thiopropionic acid)	0.06	0.3
Serin (α -Amino- β -oxypropionic acid)	0.2	0.6
Oxy- α -prolin	0.25	1.0
Tyrosine (p-Oxyphenyl- α -Aminopropionic acid)	4.5	1.3
Lysin (α , ϵ -Diaminopropionic acid)	5.8	4.3
Histidin (α -Amino- β -Imidazolpropionic acid)	2.6	11.0
Arginine (Guanidinaminovaleic acid)	4.8	5.4
Tryptophan (Skatolaminovaleic acid)	1.5	trace
Phosphoric acid (in casein)	1.94	ϕ

The caseins are insoluble in water, but soluble in bases, acids and salts. In milk they are dissolved by alkalis, especially by alkaline earths. There are, however, numerous intermediate stages between solution, which is indestructible by any mechanical means, and suspension, in which very minute portions of undissolved casein are kept in a finely divided state by means of calcium casein. Casein, in this state, has been appropriately designated by the Germans as "*Käse-stoff*," though this is not intended to determine what components take part in a solution of this kind. Such casein does not coagulate upon boiling, it is carried down by all heavy precipitates, and it cannot pass through heavy filters.

Cow casein contains 53 per cent. of carbon, 7.06 per cent. of hydrogen, 15.65 per cent. of nitrogen, 0.76 to 0.79 per cent. of sulphur, and 0.85 per cent. of phosphorus. It is at least a tetrabasic acid, and combines with 2.73 per cent. of sodium oxide, using phenolphthalein. It gives all the albumin reactions, even those of Molisch and Adamkiewicz, because the carbohydrate group is lacking, and only a weak reaction for sulphur. It dissolves quickly in boiling water and alkalis, and when digested by pepsin it yields albumoses. From these by further digestion a precipitate, called pseudonuclein, is deposited, which is in turn dissolved only after vigorous digestion. The pseudonucleins are not simple substances, but mixtures of albumoses and the pseudonucleic acids. These are rich in phosphorus (4 per cent.).

The casein of human milk is always precipitated from solution in finer flakes than are the caseins of the ruminants, and, under conditions similar in other respects, they yield no pseudonuclein on digestion by pepsin.

The lactalbumins agree in all their properties with the serum albumins of the same species of animals. The lactalbumin of cows, however, is said to have a higher limit of precipitation with alcohol than the corresponding albumin of the blood. Lactalbumin passes through the clay cell in filtration.

Lactoglobulin is found in abundance only in colostrum and has not yet been clearly defined.

The proteids of milk are specific; that is, their injection into rabbits (but less often with guinea-pigs and never with dogs) occasions in the serum of the blood the formation of a substance which causes a precipitate with the milk or the proteids of the same, or closely related, species of animals. It has not yet been determined whether this specific action derives its qualities from the group of proteids separated chemically or from some substance attached to them.

The nitrogenous extractive substances found in milk are urea, ammonia (probably only in milk already decomposed, or by absorption from the atmosphere of the stable), orotic acid; *i.e.*, a ureide with

the formula, $\text{CO} \begin{Bmatrix} \text{NH}-\text{CO}-\text{CH}_2 \\ | \\ \text{NH}-\text{CO}-\text{CO} \end{Bmatrix}$, and traces of creatin, creatinin,

hypoxanthin, and sulphocyanate. The so-called nucleons are artificial products.

Milk sugar is identical in all kinds of milk. It is as yet uncertain whether some other carbohydrate is also present in small quantities. It is probable that we have here to do with a substance that can be split off from lactomucin by acids.

Finally, milk contains citric acid, substances producing color and odor, perhaps also a trace of substances giving an iodoform reaction (alcohol or aldehyde), ferments, and alexins.

In nutrition great importance has been ascribed to the ferments or enzymes. It is a question as to whether the milk carries the ferments which are contained in the cells and blood serum, as a cell product or as a transudate. There have been demonstrated, so far: (1) *Superoxidases*, which decompose hydroperoxides by liberating the oxygen. By centrifugation they pass into the cream, where they adhere physically to the milk globules. Consequently, in fractional precipitation, they are deposited along with the casein.

(2) *Reductases*.—Milk in contact with sulphur, selenium, arsenic, phosphorus, etc., and with water, produces the corresponding hydrides. It also induces other reductions, for example, that of methylene blue.

It is, however, not yet quite certain that this latter is not caused by bacteria. It is likewise still doubtful whether these reducing agents are identical with the milk ferment called by Smidt aldehyde-catalase, which reduces methylene blue when small quantities of warm formaldehyde are present. Both pass into the cream under centrifugation.

(3) *Aldehydases*.—These oxidize aldehydes when air is admitted. Perhaps they are identical with the reductases.

(4) *Peroxidases*.—In the presence of the so-called true superoxides (H_2O_2 , BaO_2 , Na_2O_2 , etc.) or substances closely related to them (as ozonized oil of turpentine and the resinous part of tincture of guaiacum), these hasten the oxidization; e.g., of guaiacol, p-phenylendiamine, iodide of potassium, and many similar substances. It is probable that they hasten also other oxidative syntheses; e.g., the formation of indophenol from naphthol and p-phenylendiamine. They pass into the skim milk by centrifugation and in fractional precipitation are only precipitated along with the globulins.

(5) *Amylolytic Ferments*, which transform starch into dextrins and maltose. They appear to pass into the whey when precipitated.

(6) *Glycolytic Ferments*.—The action usually attributed to them should perhaps be assigned to bacteria.

(7) *Lipase*, which decomposes neutral fats into fatty acids and glycerin. It has not been ascertained how these substances, and the two that follow, act in fractional precipitation and in centrifugation.

(8) It has not yet been determined whether the decomposition of salol by milk is produced by a ferment, *salolase*, or merely by saponification. *Salolase* does not occur in the alcoholic precipitate.

(9) Whether *proteolytic ferments*, and even ferments resembling the autolytic, are present in the milk, as is *a priori* not improbable; or whether the effects attributed to these are due to bacteria alone, is no better determined than is the presence of rennin ferments.

(10) On the other hand, their coagulative effect on hydrocele fluid is certain.

Colostrum and centrifugal sediment, because of their abundant cellular constituents, contain more superoxidases and probably also more reductases and aldehydases than does normal milk. Colostrum contains peroxides even when normal milk contains none, or only traces. A more abundant appearance of the ferments known collectively as oxidases goes hand in hand with the presence of colostrum constituents in normal milk—for example, in that of women. A quantitative determination of the peroxidases in human milk can in this sense take the place of a microscopical examination. Moreover, most of the data concerning the quantitative relations of the ferments in the different kinds of milk are unreliable or worthless, for the effect of the ferments

depends on the reaction, the quantity of salt, and many other circumstances. The number of bacteria in the milk is also an important factor.

The alexins of the milk are derived in part from the transuded serum, and in part from the glandular cells. All the alexins produced in the body of the animal, either by experiment or by disease, pass into the milk. Between the amount in the milk and that in the blood serum there exists, in some cases, an invariable ratio (1:200 diphtheria antitoxin in horses). On the other hand, milk, and especially colostrum, is richer in agglutinins than is blood serum. Cow's milk has normally a marked germicidal potency against the cholera vibrio and checks the growth of the dysentery bacillus. Its effects upon the bacteria of typhoid, paratyphoid, enteritis, and diphtheria, and upon colon and milk saprophytes are, however, insignificant. Human milk is believed to check the growth of typhoid bacilli in a slight degree. On the other hand, the milk of women in the first weeks after parturition, colostrum especially, abounds in agglutinins for the blood of both men and rabbits. The hæmolysis due to staphylolysins, vibriolysins, agaricin, saponin, etc., is checked by the milk (woman's, cow's, goat's). The agglutinating agent appears to reside in the serum of the milk. Finally, the action of human milk in preventing the coagulation of cow's milk has been attributed to its property of neutralizing rennin. Goat's milk does not possess it. The absorption of the alexins of milk from the alimentary canal appears to occur only in the case of young animals and only when united with the proteids from the same species, and even under such conditions does not always follow (*e.g.*, tetanus antitoxin in the horse, typhoid agglutinin in rabbits, guinea-pigs, and cats). The influence of the digestive ferments is here important.

Milk contains, inorganically combined, K, Na, Ca, Mg, NH_4 , Fe, O, N, P_2O_5 , Cl, CO_2 , traces of Al, Si, Mn, Fl, I, and perhaps also H_2SO_4 . Milk ash contains carbonates, phosphates, and sulphates, and perhaps even iron which has arisen from organic compounds by incineration. Not all the salts, even leaving out of the question the bases united with the casein, are found in perfect solution. On the contrary, a part of these, particularly Ca and P_2O_5 , are precipitated by mechanical action; *e.g.*, by long standing, by filtration through porcelain filters, and by centrifugation. The gases, particularly CO_2 , are given off by simple standing.

Between 0.2 and 0.4 per cent. of unknown substances may be present in the milk. This, however, is assumed on arithmetical rather than chemical grounds and appears doubtful. These "residues" become numerous only in colostrum, of whose proteids (see *Lactomucin*, above) we have no adequate knowledge.

The color of milk is caused by the reflection of light from the sus-

pended particles of casein and from the fat globules. Skimmed milk looks blue because the blue rays are more strongly reflected.

The specific gravity is lowered by a high content of fat and raised by one of salts. A milk poor in fats can thus be brought to the normal specific gravity by watering it. During several hours after milking, or after being warmed, the specific gravity rises as much as 0.0015. This is due to the contraction of the stiffening milk globules.

The freezing-point depends on the concentration of those constituents which are in perfect solution. It is consequently affected by the proportion of fat only so far as the space for the molecules in solution is decreased by bulk of the milk globules held in suspension. That is, the higher the content of fat, the less the space for the molecules in solution; *e.g.*, in cream with 14 per cent. by weight of fat there remain available 85 space units; in unskimmed milk with 4 per cent., 96 space units. In like manner the other constituents in suspension diminish the space for the solution. No conclusion as to the state of the constituents in suspension can at present be drawn from the freezing-point. Its depression depends not only on the number of the molecules but on their nature, since mixed solutions act differently from solutions of a single salt. Solution and suspension, too, are states that have much in common and differ in degree rather than in kind.

The electrical resistance depends on the concentration of the dissociated molecules in solution and not on the proportion of sugar and urea. The constituents which are wholly or partially in suspension, increase by the electrical resistance their friction and cohesion. The reciprocal of electrical resistance is conductivity.

Its specific heat depends partly on the state of the fat globules. The more rigid they have become, the more heat is required to liquefy them.

The viscosity (internal friction) is a very complex property, which depends chiefly on the proportion of casein.

The reaction with litmus paper depends on the proportion of the dissolved components, especially of the phosphates and caseins. Since the presence of monophosphates alone would precipitate the casein, and diphosphates would turn red litmus paper blue, fresh milk can give only amphoteric or alkaline reactions. The capacity of the bases and acids to form combinations is measured by their ability to react with alkalies and acids, so as to produce changes of color in an indicator. For the alkali-combining power phenolphthalein and caustic soda are used. In testing the acid-combining power we titrate, using alizarin red, methyl orange, or blue litmus paper, with hydrochloric or sulphuric acid. Each of these indicators gives a different figure, the last named the highest, because the change to red follows only when all the casein has been combined with acids. All titrations are conventional. They

give other figures if the degree of concentration of the titrated fluids is changed, as when diluted milk is used. If, instead of caustic soda, baryta is employed, much higher values will be obtained, because of the precipitation of the insoluble phosphates.

On standing, CO_2 is given off and O is taken up. The acidity consequently decreases and with it the tendency to coagulation. The milk globules now rise and carry with them a liquid abounding in proteids (especially perhaps in lecith-albumins and lactomucins), in certain salts, superoxidases and reductases. The calcium phosphates, the casein, and the impurities, on the other hand, sink gradually to the bottom. By centrifugal force this separation is hastened and perfected. During this process micro-organisms pass both into the cream and into the slime. If bacterial decomposition can be warded off without boiling, the ferments contained in the milk gradually accomplish their function.

Filtration not only frees the milk from impurities and cellular elements, but removes some casein and apparently some lactomucin, even when only a dense paper filter is used. If the milk is drawn or forced through a filter of clay, or the like, the filtrate will contain only the lactalbumin, traces, at most, of a proteid (which acetic acid will precipitate), the whole of the milk-sugar, the citric acid, the coloring matter, all the Cl and K, and nearly all the Na, but only a part, greater or less, of the Mg, Ca, Fe, P_2O_5 . Superoxidases, reductases, and proteolytic ferments do not pass the clay filter; the peroxidases pass only slowly and in traces. Data for other ferments are wanting. The alexins that have been tested (typhoid agglutinins and bactericides for typhus and colon) do not pass the filters.

In dialysis those constituents in solution which have a small molecular weight, such as the dissolved salts, milk-sugar, and citric acid, are removed. (Human milk is reported not to surrender chlorine.) In protracted dialysis the alkali is withdrawn from the casein compounds and the casein is precipitated.

On freezing, a highly concentrated solution collects below while the fat rises in cream. The fat globules stiffen and such milk is easier to churn. If the milk is kept frozen for a long time its efficacy as a bactericide is increased. Albuminous flakes of an unknown nature separate out on thawing, and may be dissolved by heating.

Heating, boiling, and superheating over 100°C . (212°F .), cause fundamental changes in all the constituents. Slow heating to $70^\circ\text{--}80^\circ\text{C}$. ($158^\circ\text{--}176^\circ\text{F}$.) has nearly the same effect as rapid heating to 100°C . (212°F .) Milks very rich in lactalbumin or lactoglobulin (ass's milk, or colostrum in its first stage) coagulate on boiling, because the casein is carried down by the precipitation of the proteids, which are coagulated by heat. (The same occurs when cow's milk is boiled with 8 per cent. of egg-albumin.)

Cow's milk is the only other kind whose behavior under heat has been accurately examined. First the odorous matter, about 90 per cent. of the CO_2 , and half of the O and N are expelled. Consequently the acidity* falls about 17.5 c.c. n/10 sodium hydrate solution per litre and phosphates of the alkaline earths are precipitated. Even without the loss of the CO_2 , the phosphates are separated into soluble and insoluble; but this change is subject to reversal, in the course of time, whenever the phosphates, insoluble in water, may be held in solution by the other salts. A part of the citric acid, however, becomes insoluble by precipitation of tricalcium citrate (4 per cent. by heating to 75°C . (167°F .) during 15 minutes; as high as 32 per cent. by heating to 100°C . (212°F .) during 5 minutes). Consequently the filtrate from a clay filter contains less calcium (5–14 mg. of CaO per litre) and less phosphoric acid (11 mg. of P_2O_5); and the rennin coagulation is retarded by this and by the loss in acidity. The lactalbumin coagulates at 55°C . (131°F .) and upwards, but not completely until it has been kept a long time at boiling heat. By this process both simple and alkali sulphuretted hydrogen (mercaptan) are split off from it, and these give rise to a part of the accompanying odor. The precipitated lactalbumin forms, in connection with calcium salts, the foundation with which the casein, after its dissociation, also unites.

The last-mentioned process, the dissociation of the casein compounds into casein and its base, is also the cause of the formation of the skin which begins at about 50°C . (122°F .). At 60°C . (140°F .) the destruction of the ferments begins (including even the fibrin ferment of cow's milk and the salolase), and it is completed at 80°C . (176°F .). The same is true of the alexins; but typhoid agglutinins, for their complete destruction, require a heat of 120°C . (248°F .) during 15 minutes.

By superheating, the milk-sugar, and with it the milk, are browned and lactocaramel is developed. As in the case of casein, the decomposition is accompanied by the formation of acid products. The acidity is again increased (in human milk this occurs at body temperature). The milk now contains newly dissolved particles of phosphoric and nitrogenous decomposition products of the casein, and the rennin coagulation is strongly retarded, finally ceasing altogether. The resulting proteids coagulate at 140°C . (284°F .) A serum is then formed, and from this some of the products of decomposition may be precipitated by acidification, others only by phosphotungstic acid. The lecithalbumins are decomposed at body temperature.

The milk globules become partially fused by prolonged heating. It has not yet been shown that even a slight generation of fatty acids

* Heated milk colors litmus paper blue because of the dissociation of the salts with weak acids.

takes place, either from lecithin or otherwise. The specific gravity of boiled milk, after restoring the water lost in boiling, is lower by 0.004. Its transparency has been diminished by the dissociation of the casein compounds and the coagulation of the lactalbumin. Its freezing-point and the electrical resistance have been raised by the precipitation of the salts which were formerly carried in solution, but may be lowered again by superheating. The viscosity, after the lost water has been restored, begins to decrease above 60° C. (140° F.). Coagulation, whether effected by acids or by rennin, produces finer flakes in boiled milk, especially if this has been superheated.

The addition of water naturally lowers the specific gravity of milk and makes it more transparent. Owing to the hydrolytic dissociation of the salts, the freezing-point and the electrical resistance do not rise in proportion to the dilution. Dissociation of phosphates and of casein compounds decreases the combining power of bases (for example, pure milk, with an acidity of 6, when diluted with water 1 to 2, has an acidity of 4.4), but it likewise decreases that of acids. A part of the calcium salts is precipitated by reason of the dilution of the solvent and no longer passes through the clay in filtration. The viscosity falls and rennin coagulation is retarded greatly.

Addition of alcohol increases the acidity by reducing dissociation. In larger quantities it precipitates all the proteids and a part of the calcium phosphates.

Acids which have the power of transforming phosphates into monophosphates separate the casein from its solvent and precipitate it. Where special conditions of temperature and pressure are necessary in the one case they will be requisite in the other. Carbonic acid, therefore, causes precipitation only at pressures above the normal; at normal pressure it releases only a part of the suspended calcium phosphates. At the beginning and at the completion of the precipitation of casein we do not need molecularly equal quantities of the different acids, but more of those that are dissociated in a weaker state. The slighter the affinity of the acid employed the longer the casein remains in combination with its base. When concentration has reached a certain stage, sodium chloride and other neutral salts retard the precipitation by acids. The time of the appearance of precipitation depends on the acid employed; while with the same acid it depends on the rest or motion of the fluid, the concentration of the acids, and the temperature. The warmer the milk the less acid is necessary for precipitation, but the coagulum formed in acidified milk at boiling heat is a calcium compound of casein. In precipitation by acids, the fat, the cellular constituents, the superoxidases and reductases, the lactomucins, and the lactoglobulins are precipitated along with the casein. The sour whey, on the other hand, retains the lactalbumins, the milk-sugar, all the salts,

the citric acid, and probably all the other ferments and the transuded alexins; but this implies a thorough washing of the filter residues. Excess of acid dissolves the newly precipitated casein in the milk again. Indeed, with cow's milk, for complete solution as much hydrochloric acid is necessary as during the whole process of precipitation. Concentrated acids precipitate the casein once more as acidified casein.

In human milk the addition of acids usually causes a coagulum which is visible only with the microscope. By a slight excess of acid this is again dissolved; but, on the other hand, it coagulates more firmly by a heat of 40° C. (104° F.) and bacterial acidification. The fineness of the coagulum depends on the reaction between the concentration of the casein compounds and the abundance of chlorine alkalies, on the poverty in calcium salts, the relative amount of lactalbumin, and the character of the casein compounds.

Acid salts, among them alum, act like acids. When mineral or metallic salts are used, however, casein is precipitated as a compound with the corresponding base. Here, too, an excess of acid brings about solution.

Alkalies and alkaline salts precipitate calcium phosphates, especially when heated, and carry the casein compounds with them. When milk is heated with alkalies it is browned by oxidation of the milk-sugar and the casein is thoroughly decomposed. If ammonia is used, the milk-sugar turns red and the presence of citric acid evokes a violet hue (the Umikoff reaction). This, however, is true only with human milk for, when cow's milk is heated with ammonia, becoming slightly yellow in the process, the citric acid is precipitated as calcium citrate.

Of the neutral salts, saturation with ammonium sulphate* precipitates all the proteids, and the ferments and alexins adhering to them and to the milk globules. Half saturation with ammonium sulphate, or saturation with MgSO_4 , precipitates the casein compounds and the lactoglobulins; here, too, naturally, the milk globules are precipitated, together with the ferments that adhere to them. Saturation with sodium chloride precipitates nearly all the casein compounds. The salts of the alkaline earths (*e.g.*, Ca Cl_2) precipitate a part of the phosphates, and in this way increase the acid reaction and the acidity. With heat they too cause the milk to coagulate.

Rennin ferment causes the milk to coagulate at body temperature. In milk which (like that of women) naturally gives an alkaline reaction, coagulation will take place only when the alkaline matter which destroys all the rennin has been removed. Whether the coagulum is formed in one continuous mass or in several smaller ones depends on the concentration and character of the casein solution, as well as the concentra-

*Sodium casein with an acid reaction precipitates between 2.3 and 3.6 saturation with ammonium sulphate, yet the degrees of the concentration of casein as an acid are much more dependent than is the case with other proteids.

tion of the dissolved calcium salts and the strength of the ferment. In the first case the whey is forced out by the contraction of the cheese. The whey contains the lactalbumins, all dissolved substances, and perhaps a new proteid, whey albumin. It has not yet been determined whether the action of the rennin upon the casein solution results in a decomposition accompanied by the formation of what is called whey albumin, in association on a higher plane, or in an inner redistribution of the molecules. The transformation of the casein may even take place when it is cold and when no alkaline earths are present. The production of cheese arises from the fact that the paracasein calcium, as we may call the transformed casein calcium salts, is far more easily precipitated by soluble calcium salts than when in its original form. The more concentrated the solution of calcium salts, within definite limits, and the more free casein the casein solution contains, the more quickly the rennin coagulation follows. In diluted, boiled, or alkaline milk it is correspondingly slow. If the coagulation is proceeding slowly it can be hastened by a higher temperature (metacasein reaction).

In the stomach, rennin always coagulates the milk within a few minutes. In a test-tube both the peptic and the tryptic digestion of the milk-albumins (though not of egg-albumin) are retarded by rennin. We must here remark that it is not yet known whether rennin coagulation and peptic or tryptic digestion are only different activities of one and the same ferment, or whether they are produced by two different enzymes which are always found together. The less casein in the milk the more quickly it leaves the stomach. A large proportion of fat seems to act in the opposite way. It has not yet been decided to what extent pepsin decomposes the proteids in the stomach. According to some experiments, the milk fat is found to be almost half-decomposed in the stomach itself. On the other hand, where milk is the only nutriment, the greater part of the fat seems to be reabsorbed before it is decomposed. Sugar and salt, like water, are absorbed in the beginning of the small intestine, the former after it has been hydrolysed by lactase into galactose and dextrose. The calcium phosphates also, in limited quantities, can be taken up undissolved. By tryptic digestion the proteids are ordinarily separated into polypeptids, that is, into complex compounds of the amino-acids. The caseins yield products of decomposition more strongly acid than those from egg-albumin. Boiled milk is more quickly decomposed. Erepsin, a ferment of the mucous membrane of the intestines, decomposes casein. It attacks only peptones and never albumins in their native state.

There are but few micro-organisms which do not thrive in milk, and the majority of them produce fundamental changes. In this regard they may be classified as follows:

(1) *Those which produce Lactic Acid.*—These decompose the milk-

COMPOSITION AND PROPERTIES OF DIFFERENT MILKS.

	Woman.	Cow.	Goat.	Am.
Water	86.4	88.0	85.5	91
Total solids	13.6	12.0	14.5	9
Calorific value per gram of ash-free dry substance, in calories per litre	5.493-5.878 736-790	5.959 673	5.9 803	5.3 427-490
FAT:				
Percentage in the milk	5.0	3.4	4.8	1.0
Specific gravity at 15° C.	0.97	0.93	0.86	—
Melting point	30-34°	31-34.6°	30-35°	15-17.5°
Congealing point	19-22.5°	25-30°	31°	10°
Calorific value in calories per gram	9.392	9.318-62	9.241	9.227
Iodine value—i.e., per cent. of iodine which can combine with the fat	32-58	26-49 (33-36)	34.6	—
Saponification value—i.e., milligrams of KOH necessary to saponify 1 gram	218	213-227	226	—
Reichert-Meissl value, as measure of volatile fatty acids	2.5	27	24	9.0
Hehner's value, as measure of insoluble fatty acids	89	85-91 (87)	86	—
Per cent. fraction of volatile fatty acids	1.4	6-8	—	—
Per cent. fraction of insoluble fatty acids	49	54-60	87	—
Per cent. fraction of oleic acid	50	34-38	—	—
Per cent. fraction of cholesterol	0.6	0.5	—	—
Total nitrogen	0.15-0.25	0.55	—	—
Proteid nitrogen	0.12-0.17	0.5	—	—
Casein nitrogen	0.097	0.45	—	—
Extractive nitrogen	0.03	0.05	—	—
Casein	0.6-1.0	3	3.8	0.6-1.8
Lactalbumin and lactoglobulin	0.5	0.3	1.2	0.3-0.7
Urea	0.02	0.01	—	—
Ammonia (?)	0.018	0.02	—	—
Milk sugar	6.4	4.4	2-5	5-6
Citric acid	0.005-0.07	0.12-0.2	0.1-0.15	—
Color of the lactochromes in native serum	red	yellow	—	—
FERMENTS:				
Superoxidase (catalase)	++	+	+	—
Reductase	trace	+	trace	—
Aldehydase	0	+	trace	—
Peroxidase	0	+	+	0
Amylase	+	+	+	+
Glycolytic ferment	?	?	?	?
Lipase, reckoned from the amount of acid split off	5-7	2-3	1	1
Salolase	+	0	0	+
Proteolytic ferment	+	+	+	+
Fibrin ferment, tested on human hydrocele fluid	+	trace	—	—
Total ash per thousand	1.4-28.	7.0	7.7-10	4-5
K ₂ O	0.8	1.7	1.3	0.84
Na ₂ O	0.2	0.5	0.6	0.3
CaO	0.3	2.0	1.9	1.0
and completely soluble	0.17	0.6-0.8	—	—
MgO	0.06	0.2	0.15	0.13
FeO	0.005	0.01	0.03	0.01
P ₂ O ₅	0.46	2.4	2.8	1.5
Organic phosphorus in complete solution	0.09	0.6	0.7	0.26
Inorganic phosphorus in complete solution	0.37-80	.8-75	2.1-75	1.24-82
Cl	0.43	0.95	1.0	0.31
Gas in per cent. of volume	7-7.5	4.2-8.6	—	—
CO ₂ in per cent. of volume	2.3-2.9	3-7	—	—
O in per cent. of volume	1-1.4	0.1-1	—	—
N in per cent. of volume	3.4-3.8	2-3	—	—
Specific gravity at 15° C.	1.032	1.028-1.034 (1.032)	1.0267-1.038 (1.032)	1.025-1.034 (1.032)
Specific gravity of solids at 15° C.	—	1.3-1.4	—	—
Specific gravity of solids, less fat at 15° C. ..	—	1.6	—	—
Freezing point	-0.5 to -0.63°C.	-0.54 to -0.59°	-0.57° C.	-0.55° C.
Resistance in ohms	175-666 (434)	180-304 (204-255)	146	—
Conductivity 10 ⁻⁴	15-57 (23)	32-55 (44)	68	—
Internal friction at 15° C.	1.41-2.56	1.67-2.2	2.01-2.15	—
Reaction to litmus	alkaline	amphoteric	amphoteric	alkaline
Basic combining power per litre, in c.c. N/10 alkali	20-25	175	—	60-70
Acidic combining power per litre against blue litmoid, in c.c. N/10 acid	85	320-550	—	350
Complete casein precipitation by the addition of c.c. N/10 acetic acid per litre	170	1000	—	—

sugar, with formation of lactic acid, acetic acid, formic acid, alcohol, etc.

(2) *Those which produce Butyric Acid*.—These cause the milk-sugar or the lactic acids to ferment, with formation of butyric acid, propionic acid, hydrogen, and carbonic acid.

(3) *Proteolytic*.—These decompose the proteids until the amino-acids are reached, and, in so doing, usually coagulate the milk.

Besides these there are organisms that produce *coloring matter*; others that make the milk *slimy* (by impairing the milk-sugar, or, more frequently, by altering the proteids); still others that make it *ropy*, *saponaceous*, *bitter*, or *malodorous*. The organisms that disintegrate the fat seldom become important in ordinary milk. This classification, however, is not to be understood as strictly differentiating the species. On the contrary some which produce lactic acid (e.g., *B. coli*) also have a proteolytic action, and *vice versa*. At first, with cow's milk under ordinary circumstances, both the lactic acid producers and the proteolytics are active, but the former soon predominate. Indeed it is the *Bacillus acidi paralactici* Kozai (a streptococcus, which is believed to be identical with *Streptococcus enteritidis* Hirsch, the *Enterococcus* Thiercelin, and the *Bacterium lactis acidi* Leichmann) which, in the formation and decomposition of lactic acid, sours the milk and uses up the lævolactic acid, so that dextrolactic acid remains. At the temperature of incubation, on the contrary, the *Bacillus acidi lævolactici* is active, and this acid soon predominates over the inactive mixture of lævolactic and dextrolactic. Finally, mildew bacilli destroy these acids and butyric acid bacilli decompose the remaining sugar. Room has now been made for the proteolytic species.

By boiling milk, the micro-organisms that produce lactic acid are destroyed, but not the spores of the butyric acid bacillus (*Bacillus butyricus* Botkin). Consequently, semi-sterilized milk in the course of time falls a prey to one or the other of these agents of decomposition and poisons are developed whose nature is still unknown. According to Conn-Esten (1904), a low temperature is the best protection against the growth of the organisms which produce lactic acid.

III. MILKING AND THE CARE OF MILK

Here we will consider cow's milk exclusively.

Among the true bovines (*Bos taurus*) Wilckens distinguishes: (1) Cattle of the plains; for example, in Hungary. (2) Lowland cattle; for example, in Holland, the Holsteins, Oldenburgers, Shorthorns, Ayrshires. (3) Highland cattle, in Middle Germany and South Germany, Sussex, and Guernsey. (4) Mountain cattle of various kinds; for example, Simmenthaler (with broad foreheads), Algauer (short-horned), Zillerthaler (with short heads).

In general, Lowland cows, especially those from Holland, are dis-

tinguished by the abundance of the yield; Highland cows, particularly the thoroughbred English breeds, by the quality of the milk; Short-horns, by their capacity for acclimatization, which makes it possible to keep them in open sheds in winter. This characteristic tends to promote cleanliness in the sheds. Furthermore, in considering the utilization of crude milk we shall have to lay stress upon the fact that in many breeds the ratio of casein to coagulable albuminoids is 5:1, in others 3:1. Milk of this latter class is preferable for children.

The breed is the foundation on which the action of the fodder is based. Deficient nutrition, especially food poor in albumins, decreases the production of milk. On the other hand, with good milch cows, fodder rich in proteins increases the yield. Food that is rich in fat increases the fat in the milk, partly by direct transmission from the fodder, as may be demonstrated by the iodine index of refraction, by the melting-point of the milk fat, by the Halphen reaction with cotton-seed oil, or Baudouin reaction with sesame oil. Native fat in the fodder is more efficacious than is fat added from without, yet the so-called relishes (fennel, goat's beard, hops) are said to promote the formation of the fats. Increase in the supply of carbohydrates, accompanied by a decrease in the supply of albumin, lowers both the quantity and the quality of the milk. Fodder containing a large amount of sugar (*e.g.*, sugar beets) increases the amount of volatile fatty acids in the milk fat. Small portions of all the salts contained in the fodder pass into the milk, and by this means the proportion of chlorine, calcium, and iron can be modified. Mineral and vegetable poisons pass through the milk gland. The latter are especially dangerous when the animal itself is immune to the poison (as goats are to euphorbia, conium, and colchicum). Finally, vegetable germs of color and odor and vegetable ferments, are secreted with the milk. The milk may absorb odors from the fodder lying in the stable. Some foods (as turnip leaves, bad mash, and wet grass) may produce a diarrhoea in the animals, which, by causing a stronger bacterial infection in the milk, may give rise to severe cases of diarrhoea in children. The admixture of an astringent (calcium phosphate) with such food has a favorable effect. Feeding with garbage seems also to increase the germs in the milk. Much investigation is still needed to determine which foods produce the lactic acid and which the proteolytic micro-organisms in milk.

Up to about the eighth year the quantity and quality of milk increase; after that they fall off. The period of lactation continues for 11 months; with farrow cows (not pregnant) even 2 years. The milk of the first three days after delivery (biestings) mostly coagulates in boiling, because of its large proportion of albuminoids of the kind that coagulate by heat. It should not be sold. In the course of the period of lactation, from about the eighth month, the quantity of milk, the pro-

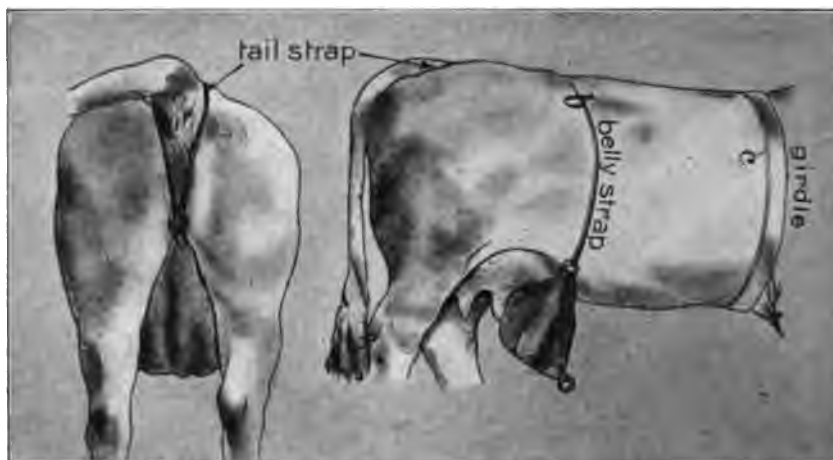
portion of sugar, the extractives, and potassium decrease, while the fat, casein, and sodium increase. In regard to the coagulable albuminoids no conclusion has been reached. With many animals heat diminishes the quantity and quality of the milk, as does the weaning of the calves and any depressing influence. Excessive work injures both, and alters the composition of the fat. The method of milking is of great importance. The quantity and the quality of the milk increase with frequency and thoroughness in milking. From the beginning to the end of the milking, whether only one or all four of the teats are milked at a time, the proportion of fat rises, slowly at first, very rapidly at the end. At a milking, for example, for successive quantities of 150 c.c. it would be 0.7, 1.2, 3, 3.9, 4.1, 4.3, 4.35, 4.35, 4.4, 4.7, 8.9. If at the close, the udders are massaged and the milking resumed (Hagelund method) a milk very rich in fat will be obtained. The bacteria are most numerous at first. The last milk may be free from germs if no infection supervenes.

The difficult problem of seeing that perfect cleanliness is observed, both in milking and in caring for the milk, is imposed on the physician. His requirements must be strictest where milk of superior quality is to be produced in special stables and sold at a corresponding price, or where a private person wishes to secure such milk for his own use without regard to cost. With unflinching tact, on the other hand, he must, in the beginning, insist on only the most necessary and inexpensive precautions in an old stable where milk of a medium quality is desired. To avoid repetition, I will state here the requirements which must be demanded in a dairy that is intended to supply milk for children:

The stable must be light, well-aired, and spacious. The more large windows the better, and they must have sliding sashes. A very useful contrivance is a glass partition at the side of the salesroom, or milking-room, so that the whole stable can be kept in view. The doors should be in two sections, so that the upper half, which should have a sash window, can be left open. The walls should be made of some porous material which is a non-conductor of heat, but admits the air (*e.g.*, wood, tuff or slag concrete, or terra cotta). They must be strong, with an air space between the outer and inner courses, and unplastered so as to remain always dry. There should be enamelled tiling or a coat of varnish for 5 or 6 feet from the floor. Ventilation outwards should be afforded by valves or tubes in the ceiling, and fresh air should be admitted as soon as the temperature rises above 15° R. (66° F.). There is ventilation of the managers at Oud Bussem, near Amsterdam. The floors should be water-proof, and covers should be provided for the openings through which the liquid manure is drained. Near the stable should be a open space for the animals to run in. This may also be used for milking. A separate milk-room like that at Ohorn Manor, for example, is perhaps the most essential improvement.

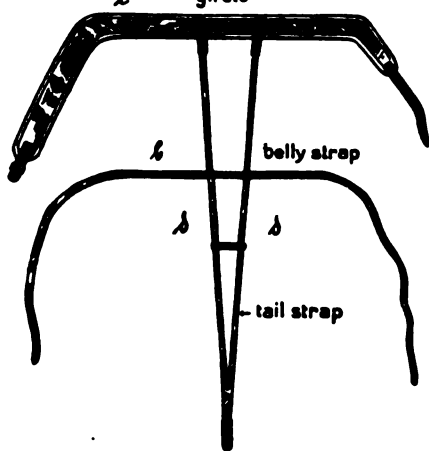
The cow's position should be such that it can be approached either from in front or from behind. This can be effected by a passage-way between the stalls. These stalls must be made of some waterproof

FIG. 23



Henkel's udder protector.

material not subject to decay, and should be provided with an automatic device for watering (F. Hüttenrauch, Apolda). The stall should be so short that when the cow is lying down its hindquarters will pro-

FIG. 24.
girdle

Henkel's udder protector.

FIG. 25.
220

Cow body protector. Figures in centimetres.

ject over the manure conduit. In order to adjust it to animals of different sizes, a pole with movable cross-bars is used in fastening the cows. In Victoria Park at Berlin the cow is separated from the manger by a reversible grating, so that it must move back after feeding.

Scattering the fodder is prevented by lattices (Hüttenrauch, Apolda), or by laths diverging as they rise, through which the cow must put its head to feed.

For litter, use turf, shavings or sawdust, straw which is not too

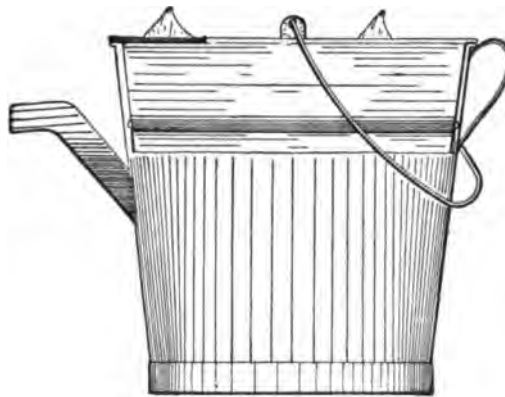
long, or straw mats (Brüssel). There must be frequent cleaning of the manure conduit, which becomes clogged by the litter. The manure should be removed either after milking or an hour before. There should be water-pipes with numerous faucets, unimpeachable water, and lavatories for the milkers. No other animals should be allowed in the stable. The health of the cows should be tested by tuberculosis inoculation before they are admitted, and they should be regularly inspected by veterinary surgeons. From time to time, the milk should be collectively tested for tuberculosis bacilli by the inoculation of guinea-pigs.

The cows must be curried and cleaned every day; their hair kept short and shaved on the inner side of the thigh and around the udder (Brüssel). The udder protector is described by Henkel. At the Ohorn Manor, near Dresden, the cow wears an apron during the milking. This is fastened by clamps and leaves the udder free. In Victoria Park the udders are cleansed with vaseline and bran. Washing the udders before milking, as is sometimes recommended, has been condemned as causing inflammation, and it is at best useful only when executed thoroughly with soap and plenty of water. Before milking the udder is usually rubbed clean with a dry or damp cloth, which is always fresh for the occasion and is carried in the milker's blouse. The tail must be tied up or washed with special care. Slapping with the tail results from annoyance by flies. After the udder has been cleaned the cow is usually tied so that it can not lie down.

The milker washes his arms and hands with soap and brush, puts on his linen blouse, and arranges the milking-stool. He should not soil his hands by further contact with the hairy body of the animal. In milking, the hands should either be dry or smeared with vaseline or lard (never with milk or saliva). The lubricant should be kept in a covered metal box and after each milking it should be melted in an oven. The first spurts should be milked on to the floor, or, better still, into a special vessel. The milker should wash anew after milking each cow.

The Andersen milking-machine is, so far, the only one that can be recommended. (Its manufacturer is Ch. Schmidt, Alt-Rahlstadt in Holstein.) Satisfactory bacteriological tests and practical experience in this regard are still lacking.

FIG. 26.



Milk pail with washable filter.

Milk into metal pails and let the cleaning begin at once. Placing a metal sieve on the milk pail apparently is not sufficient, because the jet of milk forces the impurities through. Sieves are improved by hav-

FIG. 27.

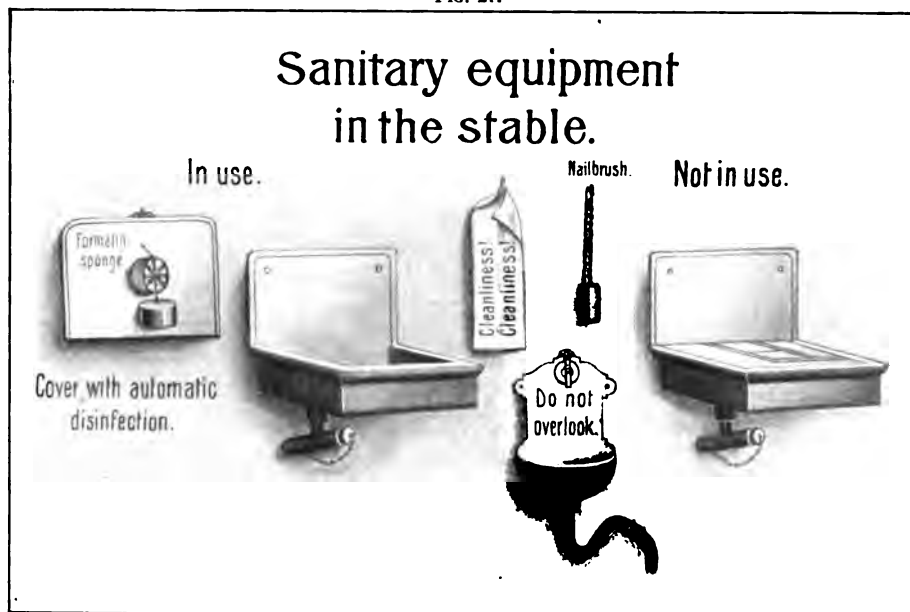


FIG. 28.



ing a layer of cotton-wool, which can be thrown away every time; perhaps also the pail with a washable strainer is preferable. Henkel would have the milk poured through the hygienic conveyer immediately after milking, passing through a cotton-wool filter outside the stable. A sponge

saturated with formalin is to disinfect the conveyer automatically. In any case, the milk is to be strained outside the stable and in a place free from dust, through fine wire sieves between which layers of cotton-wool have been pressed, or through filtering-funnels (for example, the conical milk sieve).

Then the milk should be thoroughly cooled, and at the same time aerated by running it through a cooler in which water from supply pipes, or ice-water, circulates.

FIG. 29.

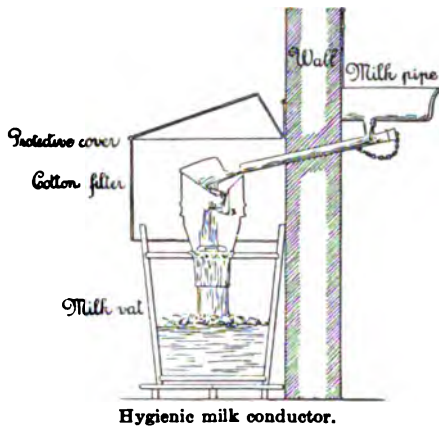


FIG. 31.



FIG. 30.

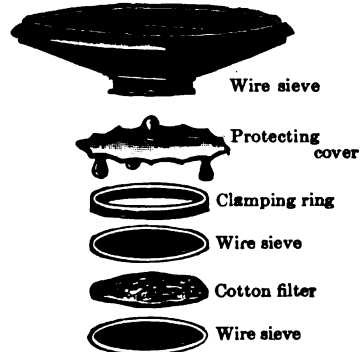


FIG. 32.



The milk should now be poured into transparent glass bottles, the cleanliness of which is a matter of prime importance. Bottles with large necks are easier to clean. Close them by metal caps or covers of waxed pasteboard.

Clean all utensils thoroughly with boiling water after using.

Although it is expensive, dry fodder is the ideal for city stables, because change of food frequently produces diarrhoea among the ani-

mals. In Victoria Park each cow receives 28 pounds of hay, 5 pounds of wheat flour, and 12 pounds of wheat chaff daily. In any case, the food should not lack variety, or be damaged or impart a bad odor to the milk (as will cabbage, ricinus cakes, or garbage), and the hay should not be exposed to rain, mildewed, or fermented. Turnip leaves or parings, swill, and draff should not be fed.

If an animal is sick, its milk should be excluded at once, and a small subsidiary stable should be fitted up to insure this.

Those who take part in the milking must not suffer from either chronic (tuberculosis, syphilis) or acute infectious diseases. Cases of scarlet fever, diphtheria, or typhoid fever in their families equally forbid their employment.

If it is desired to improve the milk produced in an old stable without excessive cost, and to obtain milk for children from certain cows, it will be well to observe the following directions:

Enlarge the windows, admit plenty of light and air, select healthy animals, and place them in the best-ventilated part of the stable. Keep these animals clean and give them individual litter. Milk them at the beginning of the milking-time and strain the milk immediately. (It is here still questionable whether several fine, unperforated straining-cloths, which can be boiled each time they are used, should not take the place of the cotton-wool filters which, according to experiments in Rodlitz, are effective only when they have been sterilized.) Finally, put the floor in good condition. In such stables I have succeeded in obtaining strippings for the use of children, though so far no attempt has been made to produce this on a large scale. The rich milk drawn at the last in milking is called strippings. After all four teats have been milked in succession, so that about two-thirds of the milk has been drawn, put a special pail beneath and draw off the rest. This will usually contain 6 per cent. of fat. Strain and cool it at once. With the aid of butyrometers and centrifugators the proportion of fat in the strippings can be determined in a few minutes, and, accordingly, the quantity of water which must be added to obtain milk containing, say, 3 per cent. of fat. For infants less than one week old the last 200 or 300 c.c. should be milked separately and enough water added to obtain milk which, with 3 per cent. of fat, has only 1 per cent. of casein and 0.23 per cent. of salts. This milk, however, must be poured at once into bottles holding but a single portion, for the cream rises very easily. This procedure requires a high degree of intelligence in the dairyman, as otherwise there will be great fluctuations in the composition of the milk.

If the animals are in pasture, all that is required is to keep them clean, strain the milk, and transport it quickly. Milk in itself has few germs. Goat's milk is often to be recommended on this account.

If the milk is not consumed where it is produced, it should—at least if used for children—be kept at 5° C. (41° F.). The other expedient, pasteurization, is an inferior substitute for thorough cooling.

Transportation in tin cans comes next. (Even the best wooden receptacles, the barrel-shaped oak ones, are difficult to clean.) The rectangular vessels of Helm (Alexander Works, Remscheid-Berlin) have some advantages. They can be placed against or on top of one another so that they mutually protect each other against heat. At the place of sale the covers can be exchanged for those provided with taps, thus rendering pouring into another receptacle unnecessary. If the milk is not to be sold directly from the cans used in transport, it is enough to have vessels that can be securely closed. Packing with cloth, paper, or straw is not permissible. Transportation by rail should be in special cars which have isolating walls filled with non-conductors of heat and

FIG. 33.



Helm's rectangular transportation cans.

FIG. 34.



Cover with faucet for the Helm can.

FIG. 35.



Revolving milk can.

which are kept cool by ice or refrigerating apparatus. For transport by wagon, cans with covers which have a cylindrical extension filled with ice are to be recommended. The cans must not be used for any other purpose and should be thoroughly washed and scalded. The Prussian law forbidding them to be washed with a solution of soda is entirely wrong.

A large part of the milk that is shipped goes to dairies, where it is either simply strained, cooled, and poured into bottles or cans, or undergoes a more extensive process.

The contents of the cans is tested by taste, or with alcohol, and is then poured through sieves into large reservoirs. (A sieve covering the entire reservoir is most effective.) The milk, usually warmed to 30° C. (86° F.), then passes through filters of cotton-wool or of gravel,

though these last need careful cleaning and are not very efficient. It is often still further purified by centrifugation. (These indeed, remove the milk slime, but the number of bacteria is apparently increased by uniform distribution.) Finally, it is cooled, and sometimes pasteurized, and then flows into the cans or bottles in which it is sold. Milk kept

over night is stored in well cooled reservoirs (5° C.; 41° F.), which is favorable to the destruction of bacteria.

It is a precarious task to prepare milk for children in such dairies, because of the promiscuous mixing of large quantities which cannot be traced backward and regulated from the start. It could be better accomplished by the separate treatment of milk from the different stables. If this is attempted the indiscriminate pasteurization of the milk does not seem expedient. In cases where the mothers have sufficient time and intelligence it would be better to have pure milk or cream, of the proper quality, delivered in its natural state and prepared at home under direction of the physician. On the other hand, there is a demand for small dairies, or separate departments of large ones, which shall devote themselves to the preparation of milk for infants, furnished in portions ready to drink, and made up, as by an apothecary, from prescriptions by a physician for each individual case. For such establishments the name of Milk Laboratories has been coined in America. Attempts at this are already found in the dairies of all large cities for commercial reasons, because children's milk opens the door of the consumer.

An ideal arrangement would be to supply the special department of the large dairy with selected milk from their own stables or from stables under their supervision. From this, and from ordinary milk for those of smaller means, the necessary mixtures could be made and put up in portions proper for a single feeding. This would require bottles much smaller than those used heretofore (50, 75, 100 c.c.). Finally, the preparations prescribed for infants could be compounded.

FIG. 36.



Cover for milk cans with ice receptacle.

FIG. 37.



Sanitary milk can.

Preparations of cream may be specified as such. These have become practicable only since the introduction of separators, since the cream obtained by gravity was more or less sour and often bitter, and even now the cream produced by centrifugation contains many more germs than does the milk. Usually the dairies give, for use with coffee, a cream with 14 to 15 per cent. of fat, 3 per cent. of albumin, 4 per cent. of sugar, and 0.6 per cent. of ash, so that by a mixture of 100 cream and 300 water and 27 Gm. milk-sugar a food is obtained similar to human milk. But this mixture cannot stand rough carriage by wagon, because butter forms easily on account of its slight viscosity. For this reason, preparations of cream in the dairies are formed with skimmed milk. A change will be effected by the machine for homogenization, in which milk or cream warmed to 80° C. (176° F.) will be forced under a high pressure (250 atmospheres) through minute channels (machines by Gaulin, Paris, Julien, Petersburg), or, under somewhat lower pressure (150 atmospheres), will be drawn between rapidly rotating disks (the Berberick system of the Deutschen Homogenisierungsmaschinen-Gesellschaft at Lübeck). The milk globules are thus made so small that under the microscope they look like fine dust. They are not entirely changed to butter, even by centrifugation, but, on the other hand, they are easily affected by the bacteria that decompose fat.

Modified Milk by Gärtner's Process.—Milk at cow heat, or 36° C., is diluted with an equal quantity of warm water that has been boiled, and is passed through a separator which is so adjusted that the tubes for the cream and for skim milk each carry off the same amount. The cream so obtained contains about 1.7 per cent. albumin, 3 per cent. fat, 2.5 per cent. sugar, and 0.35 per cent. ash, and consequently requires an addition of 5 per cent. of sugar. The proportion of fat varies with that of the natural milk.

Whey Milk by Szekely's Process.—Skimmed milk at about 60° C. (140° F.) is treated with carbonic acid at a pressure of 30 atmospheres in a strong, air-tight receptacle which can be closed and is provided with appliances for stirring, where the cheese components are mostly precipitated. Two parts of aerated whey are mixed with 1 part of cream, which contains about 10 per cent. of fat and 1.5 per cent. of sugar.

Whey Milk by the Monti Process.—Skimmed milk is coagulated at 40° C. (104° F.); the whey is freed from ferment at 70° C. (150° F.), and is then mixed with cream or milk.

Peptonized Milk by the Voltmer-Lahrmann Process.—Milk which has been heated to 100° C. (212° F.) is mixed with sugar, cream, and water as desired, is then treated for a short time at 40° C. (104° F.) with 0.5 per cent. of K_2CO_3 and a powerful pancreatin, is brought to a boil, is combined with somewhat less of phosphoric acid than is necessary to neutralize the potassium, is sterilized for half an hour at

102° C. (215° F.) and is then poured into bottles. The first heating might be omitted with advantage.

Modified Milk by the Backhaus Process.—Skimmed milk is mixed with rennet, trypsin, and 0.5 per cent. Na_2CO_3 , and kept in a kettle for about half an hour at 40° C. (104° F.); then it is heated to 80° C. (176° F.); the cheese is sifted from the whey, the latter is mixed with cream having 12.4 per cent. of fat, and is sterilized in bottles. There are 4 mixtures: (1) 25 cream, 25 water, 50 whey, 2 milk-sugar (1.5 per cent. of albumin, of which 1 per cent. cannot be precipitated by acetic acid); (2) 25 cream, 75 whey, 2 sugar; (3) 25 cream, 25 water, 50 skimmed milk, 2 sugar; (4) 25 cream, 75 skim milk. Baumann peptonizes the skim milk by papain and adds cream.

If after any of these operations it is desired to free the milk from germs, it may be sterilized in the bottles at from 100° C. to 105° C. (212–221° F.) by steam or steam-heated water. The Gerber-Wieske apparatus, with which the bottles are shaken while heating, is advantageous because the sterilization is more certain and the partial fusion of the globules, which otherwise takes place, is prevented. In the latest apparatus, cooling by water immediately follows sterilization. Sterilized milk should not be browned.

Recently other methods have been proposed for obtaining milk with few germs; *e.g.*, by the addition of formaldehyde in the ratio of from 1:10,000 to 1:40,000, by which the growth of the lactic acid germs is decidedly checked, but the proteolytic varieties, and possibly the pathogenic, are not destroyed. This addition is not without interest for infants as pasteurized formalin milk is almost germ free.

In Budde's process, milk, as fresh as possible, is combined with 0.035 per cent. hydrogen hyperoxide, kept for half an hour at 50° C. (122° F.) then for 2 to 3 hours at 52° C. (125.6° F.), cooled, and put in bottles. A total destruction of the pathogenic germs is not assured. Autodigestion of albuminous matter is probable. The Nectar Company in Paris saturates milk with oxygen, under a pressure of 2 to 3 atmospheres, in bottles which have a glass ball resting in the neck, and keeps them for 2 hours at 70° C. (158° F.) The oxygen is perhaps without importance.

Irradiation of ultraviolet light (Seiffert), the conduction of alternating electrical currents of 110 volts by means of carbon electrodes (Guarini and Samarini), and ozonization (Dorn) have never as yet been tested.

The production of buttermilk will also be a field for the milk laboratories. Yet it has not been determined whether we may rely on the antagonism of the organisms that produce lactic acid to the butyric acid bacilli and the proteolytic microbes or whether aseptic milk must be used for this purpose also. This would rob the buttermilk of its com-

mercial advantage. Ordinary buttermilk is valued at 1 cent a quart. Perhaps in the case of buttermilk both superior and ordinary grades will be produced. Since more butter can be obtained from cream that is slightly sour and granular than from sweet, most of the cream has always been allowed to sour. In spontaneous acidification the unwelcome germs of decay may grow profusely. For this reason, as early as 1894, acidifiers, either liquid or made into a powder with flour, were introduced into the market. These are, however, not always fresh cultures of the *Bacterium acidi lactici*; in fact, they more often contain the bacilli of butyric acid. The dairies propagate these for 18 to 24 hours at 30° C. (86° F.) in pasteurized skim milk, or add them directly to the cream, which has already been pasteurized and cooled to 5° C. (41° F.). This now stands for 18 to 24 hours, at from 16°–20° C. (60°–68° F.) and is occasionally gently stirred until it reaches an acidity of 24 to 30 degrees, which the specialist can ascertain by tasting. Then the cream is made into butter at 11°–16° C. (52°–60° F.) producing at the same time a buttermilk with about 91 per cent. of water, 0.5 per cent. of fat, and over 3 per cent. of albumin, having the appearance of fatty milk, a purely sour taste, without bitterness, and an acidity of 24 to 30 degrees. The casein (especially that from cream in its native state) is said to be pulpy, not granular. The usual practice of pouring cold or warm water into the churn should of course not be adopted here. Whether buttermilk made from pasteurized unskimmed milk, as recently attempted, will be equally valuable is still doubtful, as in former attempts the casein produced was far too gritty. On the whole, an accurate chemical and bacteriological study of a satisfactory buttermilk has long been needed.

The duty of physicians in regard to dairies will be restricted to determining by inspection the value and the purity of their products. Whether a milk in its course from stable to consumer has been pasteurized or sterilized once, or oftener, should be marked on every one of its products,

Milk is sold directly from the dairy in shops, by delivery, and from milk-wagons. The milk that is delivered ought to suffer no deterioration. A pasteboard cover is better than the easily removable lead. Shops in which milk is sold must be abundantly ventilated and cool, and must not open into sleeping-rooms. Stationary receptacles must, in Prussia, bear a permanent mark, visible to purchasers, designating their contents either as unskimmed milk, containing at least 2.7 per cent. fat, or as skimmed milk. Half milk, which in Austria is sold under various names as a mixture of unskimmed milk, skim milk, and water, should be strictly excluded from sale. The physician must recommend the shops which are clean, reliable, and, in summer, provided with ice. It is almost impossible to supervise the sale of milk from the primitive

wagons of the milk-women: these have rightly been supplanted by the modern milk-wagons of the dairies, which carry ice in summer and draw the milk through several faucets, which are protected by valves. The cans should be sealed in the dairy and furnished with a mixer, a kind of wooden float. Some means should be adopted to protect the wagons from the dirt of the streets. Booths where the milk is sold by the glass, as at Cologne, and slot-machines for cold and warm milk, as at Stockholm, are commendable.

Hygienic care of milk can be furthered by legislative enactment, or by corporate or private dairies which secure milk of the proper quality by paying according to the amount of fat, and which promote cleanliness by stipulating in the contract that the milk shall be tested for impurities. In this effort, rewards of stable employees for neatness, and warning and exclusion of the producer for uncleanness, may play a part. Associations of milk producers may also be formed, under the guidance of physicians and veterinarians, who should assume the direction and advice, while the members receive the benefit of a special designation for their milk. (Certified Milk in New York.) Insurance against loss by epidemics among cows or the employees may be obtained through associations or unions. Neat stables should be encouraged in the cities and unclean ones abolished. The use of skim milk and buttermilk should be promoted, in order to secure for these by-products a price corresponding to their nutrimental value. All producers should be educated in the art of cleanly milking.

[Milk is on sale in many cities of the United States which is vouched for and regularly examined by a committee or commission appointed by the local medical society. Such milk is sold as "Certified Milk" having the guarantee of the medical society. The following list of requirements of the Albany County Medical Society will give an idea of the standard of cleanliness required:

1. THE BARNYARD.

(a) It must contain no manure in Summer and none in contact with the stable in Winter.

(b) It must be well drained and kept reasonably clean.

2. THE STABLES.

(a) The ventilation and light must be sufficient for the number of cows stabled, so that the barn shall be light and the air never close.

(b) The floor shall be of wood or cement.

(c) The ceiling shall be tight, if a loft above is used.

(d) Basins, hand brushes, clean water, soap and clean towels shall be provided in the barn or adjacent dairy room.

(e) The stable shall be whitewashed in the Fall, and in the Spring if necessary.

(f) A sufficient number of lanterns shall be provided to allow the milking to be carried on properly.

(g) Clean the ceiling and sidings once a month.

(h) The bedding shall be shavings, saw-dust, dried leaves, cut straw or other material that meets with the approval of the Committee.

- (i) The soiled bedding must be removed daily.
 - (j) The manure must be removed daily from the stalls and open manure-gutter. If a covered manure-gutter is used, it must be kept in a sanitary condition.
 - (k) The application of land-plaster or lime on the floor daily is recommended.
 - (l) Sweep the entire floor outside of the stalls daily at least an hour before milking is begun.
3. WATER SUPPLY.
- Pure water must be used for all purposes. It must be accessible and abundant.
4. THE COWS.
- (a) Discard milk containing mucus or blood and that from any diseased cow, and also the first few streams from each teat.
 - (b) Reject milk from any animal forty-five days before and six days after calving.
 - (c) The food given must be suitable both in amount and kind and must not give a disagreeable flavor to the milk.
 - (d) Keep the cows clean on flanks, belly, udder and tail and groom thoroughly every day.
 - (e) Clip long hairs about udders and clip the tail sufficiently to clear the ground.
 - (f) The cows must be kept from lying down between the cleaning and milking. The best means of accomplishing this is by throat latches.
 - (g) Clean the udder thoroughly before milking with a clean damp cloth and use soap and water when necessary, and dry with a clean towel.
5. THE MILKERS.
- (a) No milker or assistant shall have any connection with the milk at any stage of its production if he has any communicable disease, or if he has been exposed to Scarlet Fever, Diphtheria, Typhoid Fever or Small-pox.
 - (b) After having everything prepared for milking, thoroughly wash the hands with soap, water and brush, so that they may be clean when milking is begun.
 - (c) The hands and teats must be kept dry during milking. If they become moistened with milk, they must be wiped dry with a clean towel.
 - (d) Suitable clean outer garments, such as overalls and jumpers must be put on before milking.
 - (e) Milking stools must be kept clean.
6. UTENSILS.
- (a) Strainers, whether metal, gauze or cotton, must be absolutely clean when used for straining milk.
 - (b) All dairy utensils must be absolutely clean and free from dust.
7. THE MILK.
- (a) The milk must not be adulterated in any way.
 - (b) It must average four per cent. of butter-fat.
 - (c) Cooling must be begun within thirty minutes after the milking. The temperature of the milk must be reduced to 55 degrees Fahrenheit within two hours after milking and 50 degrees Fahrenheit within three hours and kept below that temperature until delivered to the consumer.
 - (d) When delivered to the consumer the milk must not average over 10,000 bacteria per cubic centimetre from May 1st to September 30th, and not over 5,000 bacteria per cubic centimetre from October 1st to April 30th. If the Committee's requirements are fulfilled, the bacteria will not be in excess of the number permitted.
 - (e) All dairy utensils, including bottles, must be thoroughly cleansed and sterilized. This can be done by first thoroughly rinsing in warm water,

then washing with a brush and soap or other alkaline cleansing material and hot water, and again thoroughly rinsing. After this cleansing, they should be sterilized with boiling water or steam and then kept inverted in a place free from dust.

8. THE DAIRY.

- (a) The room or rooms where the bottles, milk pails, strainers and other utensils are cleaned and sterilized should be separated from the house, or where this is impossible, have at least a separate entrance, and be used only for dairy purposes.
- (b) All milk must be delivered to the consumers in bottles.
- (c) All bottles, after filling, must be closed with sterilized discs, and capped so as to keep all dirt and dust from the inner side of the neck and the mouth of the bottle. The labels of the Milk Committee must be applied at the dairy.

9. INSPECTIONS.

- (a) The farms which furnish "Certified" milk must always be open to inspection by the Committee.
- (b) Samples of milk will be regularly taken for bacteriological examination once a month.
- (c) Each farm furnishing "Certified" milk shall pay one dollar each month to the Committee for printing and other expenses.]

The physician must direct the straining of all milk for the use of infants through thick straining-bags, made of the finest bolting-cloth, or through cotton-wool filters for domestic use. This is at the same time a test of the impurities. Tests by tasting and boiling are also important; the alcohol test, in spite of its simplicity, will hardly find acceptance. The integrity of the covers of bottles of prepared sterilized milk can be verified by the respective adhesion and contraction of the rubber covers, and, in this case as well as when metal caps are used, by the clacking sound which is produced by tapping on the bottom of the inverted bottle with the knuckles. Cooling may be effected by means of refrigerators, by cooling-jars connected with the water pipes, by water dripping from a faucet upon the bottles packed separately in shavings, or, when there is no other way, by changing the water. A refrigerator built into the wall near the waste-pipe and cooled by the water passing out through coils of pipe encircling it would be a great convenience in every home.

Sterilization by boiling for 10 minutes in individual bottles submerged in water is to be preferred, because it insures a definite amount of food, uniform proportion of fat, and sufficient sterilization, while forming only a thin skin. The appliances for drawing the milk should be ample, and automatic covers should be provided for the bottles, as described by Soxhlet, Oldendorf, Gentile, etc. The cheapest are medicine bottles (obtainable in any size), which should be boiled in a pot packed with shavings and half full of water. The bottles may afterwards be tightly closed with corks, which were also boiled at the same time. The cooling in the pot should be first gradual and then rapid. The bottles should be thoroughly cleaned with soda and a brush.

Milk thermophores are reliable only when the bottles are still hot when introduced.

IV. EXAMINATION AND ANALYSIS OF MILK

It will not take the physician long to determine from what animal a given milk is derived. By Steinegger's method, which is given below, an addition of as little as 15 per cent. of goat's milk to cow's milk will be detected. Milk not over 24 hours old is to have its cream removed by centrifugation. Then 100 c.c. of the skim milk is to be heated to 50° C. (122° F.) and combined with 10 c.c. of 25 per cent. ammonia water, and is to be kept at this temperature and mixed half hourly. After an hour and a half it must again be centrifugated. Within from

FIG. 38.



Henkel's apparatus for estimating amount of dirt.

2 to 3 hours a precipitate of albumin will have formed in the goat's milk. One way in which human milk may be distinguished from cow's milk is by the Umikoff reaction. Heat 5 c.c. of milk with 2.5 c.c. of 10 per cent. ammonia to 60° C. (140° F.) for a quarter of an hour. Human milk turns violet-red; cow's milk, if it does not contain formaldehyde, turns yellow. With other kinds of milk the biological test is more reliable.

The Amount of Dirt.—Add 4 drops of formalin to every 100 c.c. of milk and let it stand in a beaker, or in a bottle with a graduated strainer (Gerber, Zurich); or centrifugate it either in ordinary vessels or in those described by Thörner; or strain it in definite quantities

through very fine bolting-cloth or through cotton-wool filters. The thickness of the layer of cotton-wool is very important. Either read off the height of the layer on the graduated scale or dry the refuse and weigh it. Choice milk must not contain any. For ordinary milk, from 5 to 10 mg. per litre may be allowed. Henkel's apparatus for estimating the dirt is made in the form of a filter. The tin cylinder used for introducing the milk is bent inward at the bottom so that a part of the cotton-wool layer is perfectly protected from the dirt, and this white surface (b) serves as a standard of comparison. On an ordinary tin can (c) is set a pan with a perforated bottom (d), on which the layer of cotton-wool (e) lies, and on this the cylinder (a) is placed and held fast

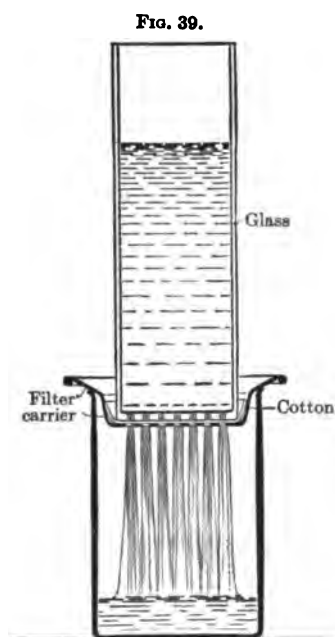


FIG. 39.
Fliegel's apparatus for estimating amount of dirt.

by a tin clamp (f). Before the milk is poured in, the perforated tin disk (g), with the strip of tin attached, is put on to prevent the layer of cotton-wool from being worn and is removed immediately after. In comparing this with Fliegel's tester, beside the protected surface of the filtering-layer, there is the advantage that dirt cannot accumulate on the bottom of the cylinder above and so cannot penetrate to the cotton. Pour in 1 litre.

Freshness and Keeping Qualities of Milk.

—Experts can judge the freshness by the taste. Milk of 11 degrees of acidity and upwards (see below) coagulates in boiling. Milk of 8 degrees or more shows coagulation when combined with an equal volume of 68 per cent. alcohol which does not give an acid reaction (the Martiny alcohol test); milk of $7\frac{1}{2}$ degrees does the same by the addition of an equal volume of 70 per

cent. alcohol. Titrate 50 c.c. in a flask, adding 2 c.c. of a 2 per cent. alcoholic solution of phenolphthalein with one-fourth normal sodic hydrate solution (Soxhlet-Henkel), using at the same time the opposite test, with the same quantity of phenolphthalein, in a flask of the same shape, without water, and on a white bottom. The titration is successful as soon as the sample turns light pink after a thorough shaking. Double the number of cubic centimetres of sodic hydrate solution is what is called a degree of acidity. Titration with NaOH of one-tenth normal strength, because of the larger amount of water, gives less than $2\frac{1}{2}$ -fold value (for example, 8:15 instead of 8:38). The limits, in normal milk, range between 7 and 8.5, but values over 8 are suspicious. Values below normal are obtained when alkalies are added and in most diseases

of the udder (for example, tuberculosis of the udder); values above normal, outside of acidification, are obtained from biestings and particularly in jaundice.

Add to 0.1 c.c. of milk 3 drops of the following mixture: 1.0 methylene blue, 20.0 absolute alcohol, 29.0 distilled water. Take 1 c.c. of this mixture to 250 c.c. of a sterilized physiological solution of common salt and keep the sample at 37° C. (98.6° F.). It should not lose its color entirely within two hours, as otherwise it is too strongly infected with vitiating bacteria.

The appearance of ammonia indicates the growth of proteolytic germs. Distil 50 c.c. of milk, 50 c.c. of neutral methyl alcohol, 10 Gm. NaCl, 0.5 Gm. Na_2CO_3 , from a 2 litre still, with 50 mm. pressure, for a quarter of an hour, in $\text{n}/20 \text{ H}_2\text{SO}_4$, and titrate. Add 10 c.c. of 10 per cent. iodine trichloride solution to 10 c.c. of milk. To the filtrate add slowly filtered 3 per cent. lime water, until, a black precipitate of nitrogen iodide appears, which is, however, dissolved by an excess of lime water.

Choice milk, when kept at 24° C. (75° F.) must show the original acidity after 12 hours, and 10 degrees at most after 24 hours. Ordinarily pure milk under the same conditions should not sour until after 24 hours.

In the *ferment test*, milk between 38° C. and 40° C. (100°–104° F.) is put in glasses that have previously been cleaned with the greatest care. The expert, after a shorter or longer interval ascertains whether the organisms that produce lactic acid, the butyric acid bacilli, or the proteolytic germs preponderate.

When a milk that has been impaired by proteolytic germs is freed from them by further pasteurization, it will disclose its condition to the expert by taste, or, chemically, by the indication of albumoses and peptones; but, this important test has not yet been worked out. A microscopical examination may be made for dead germs.

Bacteriological investigation consists chiefly in counting the liquefying and non-liquefying species. (In detail by H. Swithinback and G. Newmann, *Bacteriology of Milk*, London, 1903; H. W. Conn, *Bacteria in Milk and Its Products*, Philadelphia, 1903.) Choice milk, at the time of its delivery, without pasteurization, should contain at the most 10,000 germs to the cubic centimetre; ordinary milk has as many as 200 millions.

With most diseases of the udder, scaly particles produced by the inflammation fall into the milk; these can naturally be found in unstrained milk only. Abnormally low acidity, great fluctuations in electrical conductivity, great depression of the freezing-point, and a subnormal index of refraction for the milk serum indicate an origin from diseased animals.

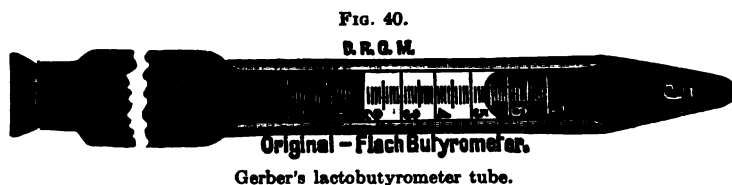
The proportion of *fat* can be determined most easily by the acid butyrometer, which shows 0.2 per cent. more than analytic processes. Into this instrument—those with the plano-convex scale are the best—is placed first 10 c.c. of sulphuric acid of 1.825 specific gravity, then 11 c.c. of well mixed milk at about 15° C., (59° F.) and 1 c.c. of the purest amyl alcohol at its boiling-point of 128° C. to 130° C. (262°–266° F.) (in the vacuum test with water there should be no liberation); close with rubber stoppers and shake the butyrometer thoroughly, after winding a cloth around it, until no coagulation is visible. Centrifugate either at once, or after the butyrometer, with the stoppers at the lower end, has been held for 10 minutes in a vessel filled with water heated to 60° C. (140° F.), in such a way that the entire scale remains under water. Read at once from the scale the percentage of fat, making allowance for the lower meniscus, while bringing the oil layer to the proper height by pressing in or screwing out the stopper. At the border line a clot sometimes separates, but, with proper centrifugation, this will be so fine that it will not disturb the reading. If you possess no centrifuge, let the butyrometer stand at 60° C. (140° F.) for half an hour and repeat the process after 24 hours.

[The Babcock fat test is the one most commonly employed in the United States. This is simple and accurate, and has replaced most of the older methods. No expert chemical knowledge is required and only one chemical is used. A small hand centrifuge, using the regulation size test-bottles and especially adapted for the use of the physician, is on the market. The test is made as follows: 17.6 c.c. of the milk are drawn into a milk pipette and allowed to run into one of the test-bottles. Smaller pipettes, holding one-fifth, one-fourth, one-third, or one-half of 17.6 c.c. are supplied when only a small amount of milk is available. Multiplying by the dilution will give the correct reading. 17.5 c.c. of clean sulphuric acid with a specific gravity of 1.082 are measured in the acid measure and slowly introduced into the test-bottle. The milk and acid are thoroughly mixed in the bottle by a rotary motion, placed in the centrifuge, and whirled for 4 minutes. Boiling water is then added by means of the pipette until the lower part of the column of fat comes within the scale on the neck of the test-bottle. A second whirling for 1 minute completes the separation of the fat. The test comes out clearer and nicer, and the fat is kept more limpid, if boiling water is put in the cups which surround the bottles. The fat thus obtained should form a clear, yellowish liquid quite distinctly separated from the acid solution.]

The proportion of *albumin* can be calculated from the proportion of nitrogen in the tannin precipitation by multiplying by 6.37, from the proportion of nitrogen in the milk by multiplying by 6, or from the difference between the calculated proportion of solids and the known

proportion of fat, sugar, and ash (about 0.71 in cow's milk). The determination from the depth of the deposit which the Esbach solution produces in skimmed milk gives only approximate values, even when centrifugated.

The proportion of *casein* is most easily determined by precipitation by alum at 40° C. (104° F.) and by the proportion of nitrogen in the filter residue. Dilute 10 c.c. of milk 4 to 6 times and keep it heated to



40° C. (104° F.); add 1 c.c. of concentrated alum solution of potassium, and then slowly add anew $\frac{1}{2}$ c.c. at a time until perfect coagulation ensues. With human milk add NaCl during precipitation, and a little calcium phosphate before filtration.

The proportion of *sugar* can be determined by polarization of milk which has been freed from albumin by mercury nitrate; by finding the reducing power of milk which has been treated according to Ritthausen



"Perplex" Centrifuge.
a.—for the examination of milk. b.—adjusted for uranalysis.

with CuSO_4 and caustic soda; or by fermenting inverted milk by Lohnstein's method (5 c.c. milk and 0.4 c.c. 25 per cent. HCl for 30 minutes in a bath of water saturated with salt, at 100° C. (212° F.). Cool and neutralize this by means of 1 c.c. liquor kalii caustici and pour off 10 c.c. Ferment this in the fermentation saccharometer with compressed yeast, at from 32° C. to 38° C. (90°–100° F.) for 2 to 3 hours. The reading multiplied by 4.33 will give approximately the amount of sugar in the milk.

The *solids* may be either determined directly or calculated from the specific gravity and the proportion of fat. (This has not been worked out for human milk.)

The *specific gravity* can be determined with a good aërometer, at definite temperatures between 10° C. and 20° C. (54°–68° F.) by plunging the clean instrument as far as the 30 mark into milk that has been thoroughly shaken, and then allowing it to float. The degrees (so the hundredths and thousandths are called; *e.g.*, 3.25—1.0325) which are thus obtained are to be corrected for 15° C. (60° F.) by adding 0.2 to the degrees of density for every degree of temperature over 15° C. (60° F.) and subtracting a like amount for every degree below. There are correction tables for this purpose. On the Soxhlet lactometer the thermometer shows the correction automatically. According to Fleischmann

$$t \text{ (the solids)} = 1.2 f \text{ (the fat)} + 2.665 \frac{100s - 100}{s}$$

when *s* denotes the specific gravity; *r* (solids exclusive of fat) is to be reckoned as *t*—*f*. There are tables for reckoning both amounts, and also the calculating machines of Ackermann (Switzerland) and Richmond (England).

When milk is *skimmed*, *f* and *t* decrease, and *s* and *r* increase.

When *water is added* all decrease, but if at the same time the milk is skimmed, *s* may remain unchanged. Watering may also be detected by the lowering of the index of refraction and of the specific gravity of the whey (with rennet whey the normal is 1.027–1.030; with acid whey—20 c.c. acetic acid to 500 c.c. milk heated to 60° C. (140° F.) in closed bottles and filtered—the normal is 1.0257–1.029), by the viscosity, by the electrical conductivity, by the lowering of the freezing-point of the milk (over—0.55° C.). If water containing nitrates is used, the nitrate reactions will furnish valuable data (diphenylamine, sulphuric acid, and HCl or NaCl with the whey; formalin and H₂SO₄).

Addition of *alkalies* may be detected by testing the milk and an equal quantity of 96 per cent. alcohol, by means of a few drops of 1 per cent. rosolic acid, producing a rosy red. It will turn brown when boiled.

Boracic Acid.—The ash when dissolved in HCl, filtered, and neutralized, becomes cherry colored with turmeric. When moistened with H₂SO₄ and decomposed by methyl alcohol, it is distilled and the vapors burn with a green color.

Salicylic acid.—A solution in ether, evaporated and dissolved in alcohol, turns violet with ferric chloride.

Formalin.—The distillate gives a dark color when tested with ammoniacal solution of nitrate of silver and liquid ammonia in a dark place. A red color is obtained by boiling with resorcin and 50 per cent. NaOH. A red violet is obtained from 5 c.c. of milk, 2 drops of diluted H₂SO₄, and 1 c.c. of solution of fuchsin which is just discolored by sulphate of

sodium. Adding a small amount of amidophenol to the whey gives a yellow color. Morphine sulphate dissolved in H_2SO_4 gives a blue ring, and there are many other reactions.

Heating above 70°C . (158°F .) can be detected by the absence of the peroxide reactions (adding guaiacol to milk and then stirring in 1 per cent. peroxide a drop at a time; fresh unheated milk turns red). Less convenient are the reactions of Storch (10 c.c. milk, 1 drop 2 per cent. H_2O_2 , 2 drops 2 per cent. paraphenyldiamine solution; gives a blue color), and those caused by a layer of tincture of guaiacum, which gives blue. When the casein has been removed by filtration (saturation with MgSO_4 or filtration by the clay cell), the filtrate, when heated to a high temperature, will give on boiling no coagulation that will not disappear with a little acetic acid.

If the physician wishes to have the compounds of the milk tested by an expert, the sample may be preserved by adding 20 drops of formalin or $\frac{1}{2}$ Gm. of trichromate of potassium to the litre.

THE FEMALE BREAST

(Anatomy, Physiology, Pathology, Technic of Nursing, Hygiene and Dietetics
of the Nursing Woman, Choice of the Wet-nurse)

BY

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Development.—The milk-glands are developed in the same manner as are the glands of the skin, and arise like these from the stratum Malpighii cutis: They appear very early in embryonic life, probably at the beginning of the third month. First, an oval thickening of the stratum Malpighii is noticeable; the accumulation of cells becomes constantly greater, and gradually takes on the form of a plump club-shaped mass, which in a later stage puts forth short compact buds, these gradually becoming longer and more slender. Toward the end of the fifth month sprouts are again given out from the buds. The first broad epithelial growth now begins to disappear, and the primary buds become ducts, which in increased numbers reach out toward the superficial layers. Through continued division of the cell ducts a constantly greater differentiation arises, which, however does not exceed a certain proportion. This stage concludes the preliminary formation of the milk-glands.

In boys and girls alike the condition remains as described, until in the latter new stimulants to growth occur from the influence of puberty. The specialized gland elements, as well as the surrounding connective tissue stroma and the subcutaneous fat, increases rapidly, this increase being greater, however, in the two components last named.

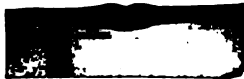
The normally developed organ of the virgin contains the corpus mammæ, surrounded by a rich layer of fat, and is composed of firm white glistening connective tissue, in which the epithelial gland constituents are so sparingly intermingled that they are not to be found in every microscopical field. The cells are found in a single layer and are flattened.

The situation is changed immediately with the beginning of pregnancy. At this time an exceedingly active proliferation commences, which differs, however, from that noted in development of the organs during puberty, in that the parenchymatous epithelial elements are most active in reproduction. The tubular glands develop new sprouts everywhere; the surrounding connective tissue, previously so poor in nuclei,

becomes richly impregnated with leucocytes and mast-cells; the active cell changes mentioned produce a corresponding manifold mitosis. Parallel with these processes is found a very rich blood supply and engorgement; the breasts are considerably increased in size and are turgescient. Under the skin the swollen veins are to be seen running a tortuous course.

At the time of delivery the development ceases. During lactation there is no further increase of parenchyma. Diligent research shows no further cell division. The completely formed organ is now either spherical or conical in form (Figs. *a* and *c*, Plate 1). The nipples are in most cases prominent, arising abruptly from the inner margin of the areola. Infrequently, to be sure, they are found less developed; and in rare cases there are no nipples, but in their place there is usually a groove-

FIG. 42.



Lens-shaped thickening of the stratum Malpighii.

FIG. 43.



Club-shaped thickening of the stratum Malpighii.

FIG. 45.



Formation of the lactiferous ducts.

FIG. 44.



Primary budding formation.

The various stages in the development of the breast. The region of the nipple is retracted; later it protrudes. The stratum corneum is shown by a thin dark line; the stratum Malpighii is dark gray; the subcutis is light gray (after Huss).

like depression, at the bottom of which the milk-glands empty themselves. This condition shows an arrested development, since the milk-ducts originally end in a shallow depression, the floor of which is gradually elevated, thus becoming the nipple.

When a puerperal mammary gland which has been hardened in a 10 per cent. formalin solution is opened by a median sagittal incision, on the inner surface will be seen the prominent true gland bodies, the parenchyma islands, surrounded by subcutaneous fat and divided by connective tissue septa, which become broader as they approach the nipple, uniting behind it (in well developed bodies) to form a thick knot of connective tissue. These knots vary greatly in size, and may be so large that the acini appear to be attached as appendices to them.

The supporting stroma contains lactiferous ducts, ten to fifteen in number, which in the central portion of the fibrinous knot described above become widened into an ampulla of a few millimetres in diameter

FIG. 46.



FIG. 47.



Median sagittal section through two lactating breast-glands. Parenchyma black, connective tissue white, fat red.

FIG. 46. Patient 26 years of age, II-para, died on the ninth day of pulmonary embolism. The child was nursed until the death of the mother. This breast shows considerable connective tissue but a small amount of parenchyma.

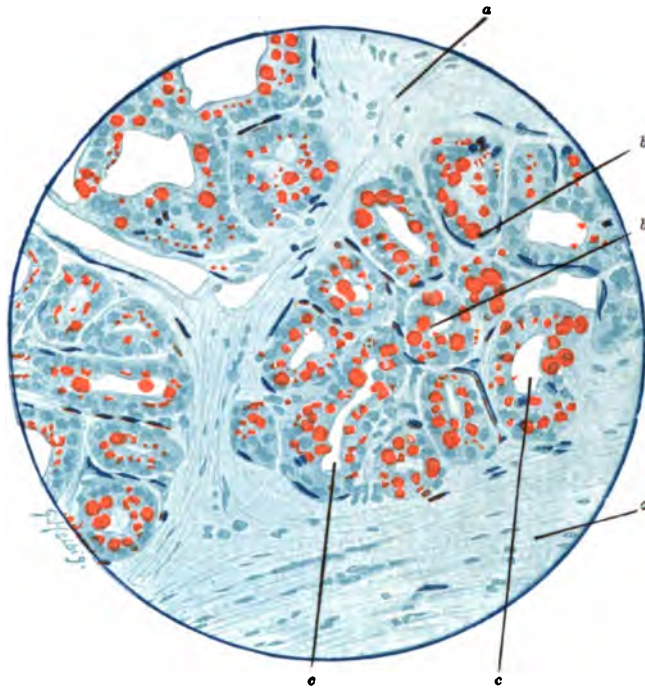
FIG. 47. Patient 32 years of age, I-para, died on the eleventh day of sepsis. Shows small amount of connective tissue but a rich parenchyma. Among the fibrinous knots behind the nipple lies the sinus galactophori, in which the milk fat, colored red, is seen.

(Fig. 47.), separated for the most part, which then empty at the top of the nipple. Each of the lactiferous ducts corresponds to a particular

gland, so that these ten to fifteen completely separate and similar bodies compose the organ.

By the nature of their structure the milk-glands belong to compound tubular glands. The peculiar secreting tubular glands are covered with a single layer of epithelium; their appearance, however, varies markedly with the condition of fulness and the phase of secretion. Sometimes the cells are low and quite flat, with a fairly homogeneous protoplasm and a single nucleus; others are cuboidal, and filled with fine fat globules; at other times they are very high, projecting into the lumen of the alveolus, thickly laden with globules of fat of various sizes, and

FIG. 48.



Section through the breast-gland of a I-para who died on the tenth day post partum. Stained with hæmatoxylin-sudan III; nuclei blue, fat red. *a*.—Interstitial connective tissue. *b*.—Intracellular fat globules. *c*.—Gland tubules.

many times possessing two or three nuclei (Fig. 48.). In the lumen of the alveolus itself drops of fat are noticed, and also occasionally the remains of nuclei. The diversity of the histological appearance depends upon the secretory activity of the glands. The alveoli with the flattened epithelium free of fat belong to the resting stage; they have already thrown out their secretion. The others have approached more or less to the acme of their function. It is to be noticed that these various conditions are found close together in a single area, and consequently the same state of secretion is not found throughout the same gland.

Formerly it was held that the microscopical examination of the

secreting glands showed that they were undergoing fatty degeneration, and that therefore the milk was produced in reality by the destruction of the gland elements. This theory of the milk being a liquefaction of the breast-gland has been prominent for several years past and is even found to-day in a number of text books. It must now be abandoned, however, since all recent researches have shown that it neither has an anatomical basis nor is supported by physiological chemistry. The milk is formed, not through a destruction of the gland cells, but through their specific secretory activity. Frequently, perhaps, there is a destruction of the superficial portion of the cells; this process, however, is of secondary importance.

The activity of the glands begins with the production of the colostrum, the clear, yellow, mucus-like, tenacious fluid, which is characterized morphologically by the presence of the colostrum bodies, which are fat-laden leucocytes. It is especially distinguishable in its chemical composition by a large amount of free coagulable albuminous bodies, and by a fat which contains an increased amount of unneutralized fatty acids, and which resembles very much the body fat, so far as its constituents are concerned.

In the last months of pregnancy drops of colostrum can be pressed out with considerable ease. At the time of delivery the production is greater, and in the course of the next few days the secretion gradually takes on the characteristics of milk,—there being, however, no particular definite moment at which one can say that the milk comes in. This usually occurs from the second to the fifth day post partum. The breasts become tense and hard, and subjectively a feeling of tension arises; ordinarily there is a slight rise in temperature (milk fever). With a constantly increasing flow of the milk stream the secretion soon loses completely its colostrum character.

So far as the origin of the milk is concerned, as has already been pointed out, it is formed through the activity and not through the destruction of the gland elements. The necessary material must be taken up through the blood. The more definite processes for the production of the principal constituents of milk—albumin, fat, and sugar—have not yet been clearly determined. The most important facts and hypotheses are here quoted.

Albumin.—Casein, the special albuminous body of milk, differs from the albumin of the blood by the presence of phosphorus. One conclusion is that the nucleic acid, derived from the cell nuclei of the glands, unites with the blood serum to form casein. In this way casein can be produced by laboratory methods with all its typical characteristics (rennet coagulation).

Fat.—Milk fat is formed from previously formed fat, the source of which is, without doubt, the fats taken up in the nourishment of the

mother and the deposits of fat already in the body. The part which the fat of nourishment plays in the formation of milk may be proved beyond question; that of the body fat may be determined in an indirect way. The material which has been brought to the glands is there prepared for the specialized milk fat.

Sugar.—Our information in regard to this constituent is very uncertain. Milk-sugar, the characteristic carbohydrate of the milk, is not found already formed in the body. Probably fermentative processes are concerned in its formation.

Composition of the Mother's Milk.—The composition of the mother's milk is constant. In the later lactation period the albuminous content is diminished. The fat content constantly and uniformly increases during the process of nursing. If the results of systematical analyses of the daily milk are collected, it will be found that the fat content approaches a constant quantity. The distribution of the individual constituents of the dry substance is, according to our analyses:

Albumin	1.29
Fat	5.01
Sugar	6.98
Ash	0.21

The albumin is represented for the most part by the characteristic phosphorus-containing casein, which is distinguished from that of cow's milk by the fine flocculent rennin coagulum. This difference exists only in the test-tube reaction. In the stomach the casein of cow's milk is broken up into a finer coagulum than in the test-tube. Undoubtedly the remainder is made up of the so-called free albuminous bodies, globulin, albumin, opalisin.

A fact of importance is that the fresh milk has physiological properties;—that it contains an oxidizing, fat-splitting enzyme, and even other properties which are destroyed by boiling.

The Beginning of the Activity of the Glands.—The mammary glands belong to those peculiar organs which functionate only periodically. The question now arises, What is the special stimulus which starts the secretion? Without considering the many more or less impossible theories, that of von Halban deserves the greatest consideration. According to this author, the expulsion of the placenta, which during pregnancy has a proliferating and tonic influence upon the breast-glands, gives rise to the secretion. The formation of the milk depends without doubt upon the influence of the nervous system, but the more intimate connection has not yet been determined.

What Starts the Secretion?—The milk-glands are stimulated to activity by reflex action. The necessary stimulus is the act of nursing. In the first days, only a small quantity, and that colostrum, is secreted; rapidly the daily amount increases, until after a few weeks the acme is

reached. The secretion remains in this condition, with slight variations, for months, almost always adapted to the needs of the child, which thrives for a long time on the same quantity of nourishment. The absolute requisite for the proper normal condition is that the act of sucking should affect the breasts amply and continuously. If this is unsatisfactory, when for example the child is weak or the mother is in poor condition, there is either a lack of development or a premature cessation of the supply of milk; only by diligent application of the child to the breast is it possible to secure the best that the breast is capable of producing.

The functional capacity remains at its height only when the breast is regularly and completely emptied at least four or five times a day. Obstruction should by no means be allowed to occur. As soon as a stasis in the milk stream occurs the phenomenon of involution sets in, which manifests itself by the immediate appearance of colostrum corpuscles (described above as fat-laden leucocytes), which return the fat from the glands back into the circulation. After nursing has been discontinued for a few days the secretion practically disappears; frequently, however, it happens that even after a longer pause it is possible to secure the return of the secretion.

Evacuation of the Milk-glands.—The complete emptying of the milk-glands is procured only by the nursing child; no other means can secure this result, as is possible in the cow. It is impossible either with the breast-pump or by the most thorough manipulations. Often a strong child nurses easily even after the pump or the hand is not able to secure another drop. The breast-pump is especially inefficient, but like all mechanical contrivances it has the advantage of relieving one of the necessity for personal dexterity. By proper manipulation with the hand it is very often possible to relieve the breast to a great extent. One must remember, however, that it is unnecessary to compress the whole breast; it being sufficient to manipulate that part covered by the areola, at the same time drawing the whole breast forward and downward, ceasing momentarily, then repeating this manœuvre. The effect is, as a glance at Fig. 47 shows, that the galactiferous sinus is first emptied, then with relaxation of the pressure is again expanded by the action of the surrounding connective tissue, and draws the milk from the more remote parts. This procedure is aided by the general compression of the organ, which is produced by the tense skin in drawing it forward. Individual limitations are very great, of course, and many women can press out the milk far better than can others.

Individual Differences in Function.—The deviation from the normal type in the gland function is of relative frequency. It is of importance to know that only slowly and with difficulty can many breasts be brought to their highest point of activity. In these cases it is neces-

sary to have a some strong and continuous nursing stimulus in order to secure the result. Again, it is significant that many breasts give out their milk more easily than do others, and that this occurs with the slightest exertion of the child.

A good breast yields for months at a time from 1000 to 1500 Gm. (1 to 1½ qts.) of milk daily; in many women, however, it is very easy to increase this production through adequate measures, such as the application of more children to the breast, and by increasing the amount of nourishment. In our hospitals, for example, we have numerous wet-nurses who daily secrete from 3000 to 4000 Gm. (3 to 4 qts.) of milk, or even more, and can therefore care for a large number of children. On the other hand, there are often women whose breasts secrete much less, and this secretion must be stimulated by diligent nursing; so that if the milk is not sufficient for the nourishment of the child, it will be a desirable addition to the mixed nourishment.

Menstruation; Pregnancy.—Considering the close relationship which the secretion of milk bears to the germinal glands, it is to be expected that the return of menstruation, or the occurrence of pregnancy, both of which are usually absent during lactation will cause some effect. As a matter of fact, the secretion is very apt to become less in either case, but this is soon followed again by an increased functional activity, if nursing goes on without interruption. There is no change in the quality of the milk, and no ill effect on the child, and therefore no reason for weaning the child. This is especially true in the case of menstruation, which with absolutely no foundation has been considered by Laien and many other physicians a signal for weaning. As regards pregnancy, it is advisable to wean the child, since the disturbances are not always absent from pregnant women, and observation has shown that children nursing from mothers who are pregnant are often not well nourished.

The duration of lactation is seemingly unlimited. So long as the mother continues nursing, so long will the secretion continue, and it can be continued for years. If other nourishment is increased, however, there is less demand for the breast-milk and it consequently decreases.

Ability to Nurse.—In the last ten years a most pessimistic idea has been prevalent in regard to the ability of women to nurse their children, but it is now recognized that the idea that the woman of to-day has not the ability to nurse is fallacious. In energetically and systematically conducted lying-in hospitals all mothers can nurse their babies equally well, and for the most part women are not prevented from nursing their children by any inability to do so, but for a variety of other reasons. Social conditions, housework, etc., are allowed to interfere, or other more or less foolish reasons, such as the very trivial

considerations of sociability and pleasure; or the mother considers herself too weak, or too nervous, or she fears she will become so fleshy as to lose her figure. On many points she is poorly advised in numerous ways, particularly by badly informed midwives.

It is the duty of the physician above all things to disseminate knowledge of the laws of health. While it is true that in the deeply rooted superstitions of former times the number of contraindications to nursing was legion, one is able to assert at the present time that there is no condition which absolutely forbids it. The general health of the individual as such, and not the presence of this or that disease, must decide the matter. The presence of tuberculosis is perhaps the only absolute contraindication, and it might be advisable in general so to consider it. Sometimes, however, upon close examination of all the conditions it might appear best to allow even a tuberculous mother to nurse.

Special considerations should be given to syphilis, in order to correct the very widespread and momentous mistakes. Two possibilities may arise: (1) the mother is syphilitic; and (2) the mother is free from syphilis, the child being inoculated by the father alone. In the first case it is easy to understand that to forbid nursing would be without reason. As regards the second, we would refer to the law of Colles, which is accepted without question. This law is, that the mother of a child which is infected with syphilis by the father is herself immune. It would be inadvisable also to prevent the mother from nursing the child in those cases where it is of special importance to provide it with the natural nourishment, and where the taking of a wet-nurse is out of the question (see chapter on Syphilis, by Hochsinger).

All other diseases, such as acute febrile and infectious affections, are not absolute contraindications. Usually it is possible to continue the nursing without injury to either party.

But above all, the often expressed fear of the mother that her strength will be insufficient must be overcome. In cases where the appetite and the amount of nourishment are sufficient to make up for the loss resulting from the milk secretion, nursing will in no way affect adversely the general condition of health, but will more likely be beneficial. Indeed, it is a well-known fact that nursing women usually thrive, and even take on more fat than they themselves wish. And in proportion as the general condition improves, they lose such ailments as were present in the beginning, such as backache, dizziness, feelings of weakness, etc.

Technique of Nursing.—The first preparations for nursing should be made early in pregnancy. Since good nipples are a condition *sine qua non* for successful nursing, the care of the nipples should begin in the last weeks of pregnancy. Moreover, since only the prominent nipples are the most desirable (Plate 1), we endeavor to improve all flat or

depressed nipples by systematic suction or manipulation; with depressed nipples our efforts are most liable to be unsuccessful, however. If the skin of the nipple is very tender the condition may be bettered by cold sponging, or by the application of tannin-alcohol (see recipe in the section on Diseases of the Breast, below).

The greatest technical difficulties are to be overcome in the first days after birth. At this time the application of the child to the breast is especially trouble-

some, not only because the mother is inexperienced, but also because it is necessary for her to remain in the uncomfortable dorsal position on account of the pelvic organs. She must give the breast to the child lying down, while she turns a little to one side with some assistance. Later she nurses the child in a sitting posture, and to best advantage on a low seat, so that the hand that holds the head of the child rests upon the elevated knee. The other hand seizes the breast in supination, so that the thumb rests upon the upper surface. The thumb is then able to keep the breast far enough away from the face so that the child's nose is left free for breathing. Only in this way is it possible to secure undisturbed nursing.

When lactation is completely developed it is only necessary to see that the breast is completely emptied and that no stasis occurs. In this connection, the mother should be warned not to nurse the child at random from either breast, so that it takes a little from each but does not empty either completely. Where there is a moderate amount of milk which is sufficient for the nourishment of the child, and this is usually

FIG. 49.



Mother nursing. The left hand holds the child's head high and is supported by the right knee, which is raised, the mother sitting on a low chair. The right hand presses the breast away from the child's nose.

the normal condition, it is especially advisable to allow the child to nurse as a rule at only one breast. Only in the first weeks, when lactation is not yet at its height, would an exception be made to this. If in spite of this it happens that there is an engorgement of the breasts, due to the fact that the child does not drink as much as usual on account of some slight disturbance of the digestive tract, the breasts must be emptied artificially. The most comfortable way of doing this is to put another child to the breast, but this method can not always be resorted to; then nothing remains except to use the breast-pump, or (what I prefer), removal of the secretion by manipulation.

Usually no difficulty arises in weaning, since lactation disappears gradually. Restoration is seldom accelerated by the numerous favorite remedies. The difficulties arising from engorgements of the breasts are overcome by the application of breast-binders and by light massage with applications of oil.

Hygiene of the Nursing Woman.—The mode of life of the nursing woman should hardly be different from that of any other woman. The clothing should be so designed that there is no pressure upon the breasts, and that these may be easily exposed. The clothing approved for the wet-nurses employed in the Infants' Home at Dresden (Plate 2, Figs. *a* to *f*.) allows plenty of room for the breast, which can be easily exposed, and when closed it gives sufficient protection to the mother.

Bodily movements and a moderate amount of work not only do no harm, but are necessary for a good general condition. The anxiety about physical changes is very much exaggerated, since neither the quantity nor the quality of the milk is affected in such conditions. Moderate sexual intercourse may be allowed, as forbidding it would not prevent it.

The breast itself must be kept warm and clean. Before and after the application of the child the nipples should be washed in sterile water or with weak boracic acid solution. Special warning should be given against the obnoxious and dangerous habit of moistening the nipples with the secretions of the mouth.

Dietetics.—Many old and, unfortunately, only too deeply rooted notions in regard to the rules for the nourishment of nursing women should be abandoned. In earlier times it was honestly believed, apparently on account of certain characteristics (*e.g.*, their white color) that soups, gruels, and broths of all kinds were especially suitable for the production of milk, and while these unattractive foods were recommended to the nursing woman, appetizing and highly spiced foods of various kinds were forbidden, since it was feared that they might have a harmful effect on the secretions. This regime is not only unnecessary but directly harmful, since the monotony and lack of flavor spoil the appetite.

PLATE 1.



a



b



c



d

a and *b*. Front and side view of a good secreting spherical breast. From a 23-year-old primipara secreting about 42 ounces of milk daily.

c and *d*. Front and side view of a good secreting conical breast. From a 21-year-old primipara secreting about 36 ounces of milk daily.

A nursing woman may eat whatever she pleases. Unprejudiced observations always go to show that the production of milk is in a large measure independent of the nourishment taken. In regulating the diet of a nursing woman it is only necessary to remember that the body loses daily with the milk about one litre of fluid and 750 calories of heat, and that this loss must again be made up; the diet should be as abundant and tasty as the nursing woman is accustomed to take. The great need of fluids must not be forgotten. Cow's milk is especially recommended as a drink.

CHOICE OF A WET-NURSE

The choice of a wet-nurse by a physician is a difficult and responsible task, which unfortunately is underestimated. Numerous conditions are to be considered, in order to do what is proper from both an ethical and a hygienic point of view. The greatest care must be taken in this investigation, because many important things (*e.g.*, syphilis) are often difficult to discover, as it is impossible to depend upon the statements of either the wet-nurse or the person who recommends her. Neither tells the truth; both have an interest in hiding many defects and bringing into prominence the desirable qualities, the one in order to secure her commission, the other to make sure of employment.

The following rules should be rigidly followed: (1) The whole uncovered body must be examined. (2) An examination of the child of the wet-nurse must be insisted upon. In relation to this point one must always be especially careful that some other child is not substituted.

Make sure that the nurse is free from vermin. Their presence need not discourage one, providing the nurse is otherwise acceptable, especially since good wet-nurses are very difficult to secure, but one should use antiparasitics systematically and energetically. Nor should we allow ourselves to be prejudiced by superficial blemishes (defective teeth, emaciation).

After settling these less important points our attention should be directed to a careful examination of the body, in order to determine whether the nurse answers to the two requirements which need not necessarily be demanded of the mother but which must be absolutely insisted upon in the nurse: (1) She must be strong and well and free from all contagious diseases. (2) She must possess a sufficient supply of milk.

In relation to the above points I might state, by way of illustration, that a mother suffering from epilepsy can often nourish her child, while the disease in a wet-nurse would be an absolute contraindication. Important distinctions exist in relation to syphilis, which will be noted later on.

After a general idea of the physical condition of the wet-nurse who applies has been secured, attention should be directed above all else to the uncertain symptoms of tuberculosis, syphilis, and gonorrhœa. These three infectious diseases are so wide spread that they are of very great importance in the examination of the nurse, since the child is especially susceptible to inoculation by the virus of these diseases.

Tuberculosis.—Frequently the previous history will give us a sufficient clue. Beyond this, one should examine for scars at the usual sites of tuberculosis in children,—the region of the glands of the neck and about the joints. It is hardly necessary to call attention to the importance of the auscultation of the apices of the lungs. Lighter grades of tuberculosis, which are not apparent clinically, may be detected by the tuberculin reaction. Injections should be made every other day, if no reaction occurs, of one to five milligrams. The temperature should be taken every two hours. The reaction is positive if the temperature reaches 0.5° C. (0.9° F.) higher than the maximal temperature of the previous day.

If one were to exclude from such duties all women who show this reaction it would mean that about a third would have to be declared useless. It is not necessary, however, to be so rigorous, as inactive encapsulated colonies give a reaction, even though there is no danger of infection. It is only necessary to exclude those wet-nurses who give clinical evidences (crepitation at the apex) at the time of the elevation of the temperature.

Syphilis.—In regard to the special signs of syphilis, it may be necessary to consult the special text books on syphilology. Here we can only notice especially important points. Above all, the child of the wet-nurse should be examined, for it often shows characteristic lesions even when the mother has none or at most only suspicious symptoms. The pharynx of the applicant should be examined for perforations and scars of gummatous processes, papules in the mucous membrane of the mouth, and specific tonsillitis, which shows itself as a sharply defined redness. On the neck we find the leucoderma of syphilis; on the buttocks an extensive exanthem. The anal and genital regions should be examined for condylomata lata. Lastly, the lymph-glands are examined. Multiple indolent swellings, especially of the inguinal, cubital, and cervical glands, are always suggestive. A verified suspicion of syphilis renders a woman unfit as a wet-nurse.

As little as one desires to have a syphilitic wet-nurse for a healthy child, it is just as undesirable to give a syphilitic child to a healthy nurse, and it should be considered a crime to make such an arrangement knowingly. Allusion to these conditions unfortunately appears not to be superfluous, since primary lesions of the nipples in wet-nurses are not uncommon.

Gonorrhœa.—Gonorrhœa is only satisfactorily diagnosed by the presence of the gonococcus. The secretion from the cervix, as well as that of the urethra, must be carefully examined. A single examination often leads to a mistaken diagnosis.

Because of the great importance of syphilis and of gonorrhœa, and of the difficulties of diagnosis, especially of the former, consultation with a dermatologist should be considered.

EXAMINATION OF THE BREASTS

After a consideration of the general status we should proceed to make a special examination of the breast and the milk. A microscopical and chemical examination of the latter need not be undertaken, since it neither gives important results nor appears to show, especially in a practical way, the significant peculiarities in the composition of the milk. At the most, the presence of the colostrum corpuscles would show that the breast was engorged.

The only real object of the examination is to get an idea of the functional capacity of the glands. But just as it is difficult in a single examination to reach a satisfactory conclusion in regard to the general physical fitness of the wet-nurse, and this indeed forms the foundation of one's decision, so is it absolutely impossible to determine the quality of the milk. Neither the form nor the size of the breast is decisive. It is customary to consider the conical breast more productive than the spherical; but this rule has a great many exceptions. Nor can the size of the breast influence one's judgment. The accumulation of subcutaneous fat around the central mass of fibrous connective tissue may be the chief cause of the greater dimension of the organ, and palpation is of little avail, because though one may feel the lobular gland tissue he cannot know how thick the stratum is. As a matter of fact, those breasts which contain a rich parenchyma will be less resistant than will those which have considerable connective tissue. In this connection it is well to remember that the nurse to be examined, in order to deceive, may not put the child to the breast for a long time, so that the full breast will be larger and firmer than normal. The ability to produce a stream of milk soon after the child has nursed, which has so often been pointed out as significant, means very little.

In short, external examination, palpation, and efforts to produce a flow of milk are not sufficient to prove the productiveness of the breast, even when examined by an expert. The only suitable method is by weighing a child, preferably that of the nurse, both before and after it has been nursed. If this is done with suitable scales, for a sufficient time (at least a day), it forms an absolute objective proof of the functional activity of the breast and the amount of milk produced, without possibility of error.

In order to avoid overlooking a single factor, it is best in examining a wet-nurse to follow a certain scheme, something like the following:

The entire body must be exposed.

I. Anamnesis.—When was the child born; how many other children; miscarriages (syphilis); ancestry (tuberculosis).

II. Examination of the whole body.—Vermin (hair of head and pubes); skin, chest, and abdominal organs.

III. Infectious diseases; namely:

Syphilis.—*Pharynx*: Perforations, scars, angina specifica. *Neck*: Leukoderma. *Buttocks*: Exanthemata. *Anus and genitals*: Papules (condylomata lata). *Lymphatic glands*: Cervical, Cubital, Inguinal. Gonorrhœa.—Examination for gonococci from cervix and urethra.

Tuberculosis.—Scars from childhood. Cervical glands. Articular surfaces of bones. Auscultation of apices of lungs. Tuberculin reaction.

IV. Examination of breasts:

Nipples.

Quantity of milk.—(a) Estimation according to form, palpation, and production of a flow of milk (possibility of engorgement to be considered). (b) Accurate proof of function by nursing.

V. Examination of children of wet-nurse (beware of deception):

Condition of nourishment.

Syphilis.

An indirect conclusion may be drawn as to the suitability of the wet-nurse by the appearance of her child. If it is well nourished, with a weight and development normal for its age, it is proper to assume that the foster-child will find sufficient nourishment. It is nevertheless well to remember, in all conclusions which are based upon the condition of the nurse's child, that a strange child may be shown, a deception which is frequently encountered. We must therefore not depend too much upon impressions obtained in this way.

It will be seen from the foregoing how many difficulties have to be overcome in the examination of a wet-nurse. If, however, the conditions are such that it is impossible to make use of the suggestions above recommended, we must do the best we can with the means at hand, making the prognosis with the greatest caution. If, on the other hand, it has been proved that the person presented is healthy and has an abundance of milk, it is well to be satisfied and not to make unnecessary further demands.

It has been shown that there are numerous conditions of lesser significance, which need not be considered. It is necessary, however, to repeat the views concerning the significance of the age of the milk, which are widely held in medical circles, and which lead to unjustifiable diminution in the number of available wet-nurses. It is not true that the child of the wet-nurse and the child to be nursed must be the same age, that "young milk" is suitable only for a young child and "old milk" for an old one. The theoretical arguments which Bunge has brought forward in the matter have been proved false in practice. His views concerning natural nourishment, the ability to nurse, etc.,

PLATE 2.



a



b



c



d



e



f

Costume of Wet Nurses in the Dresden Säuglingsheim.

valuable as they may be, have often led to wrong impressions. On behalf of the practicing physicians who make a specialty of children's diseases, such pessimistic ideas, particularly in regard to the ability of women to nurse, which results in the non-use of wet-nurses, must be energetically opposed. In the Dresden Nursing Home we are accustomed to give the weakly premature children to those nurses who have lived for some time in the house and who are accustomed to a careful technique. In spite of the noticeable differences in ages, the children thrive splendidly. If then it becomes necessary to provide a nurse for a newborn child, which is most frequently the case, do not be concerned if she has already nursed a child several months. If there is nothing else against her, she will certainly fulfil the necessary requirements.

THE FUNCTIONAL CAPACITY OF THE NURSE AND NEEDS OF THE CHILD

In the choice of a nurse, the first thing to consider, as has already been pointed out, is whether or not there is a abundant supply of milk. Sometimes, and that not seldom, this overabundance may lead to faulty conditions, which are not likely to be discovered without complete knowledge of the subject, and which may lead to abnormal conditions of the nurse or the nursing.

In the usual run of cases it often happens that a newborn child which needs very little nourishment, or a sick, weakly child, is given to a nurse who has full lactating breasts. In either case the child is not able to empty the breasts, and if no other measures are taken the inevitable result is a stasis and a loss of the gland function. The nurse is dismissed because she "has no milk." But if care had been taken of the productive functions of the glands, things would not have come to such a pass. The child would have had a good foster-mother, and the latter would not have been deprived of her milk. Wrong has been done to both parties.

The possibility of such an occurrence must be kept constantly in mind, so long as the child, for either physiological or pathological reasons, does not take sufficient nourishment. In such cases it is necessary to empty the glands with the breast-pump or by massage. I would advise, as the most important and best means, the additional application of the nurse's own child; which, for ethical reasons soon to be given, cannot be urged too strongly. By this means the child is able to continue nursing.

On the other hand, it may easily happen that the child drinks more than is desirable for its age, when the wet-nurse has nursed for a long time, since the milk then flows more readily. If we are not able to regulate the amount of the meal by means of the scales, the anomaly will first be noticed when any digestive disturbance appears. It is then often

argued that the milk of the nurse is not suitable, while the indisposition is only due to overfeeding.

From such facts we might learn that we should never fail to determine definitely by weight the amount taken by the child at each feeding.

I cannot refrain from bringing up two points, even though I know that the regard for ethics and social justice will usually prevent the physician from making such mistakes. It is well to remember that when the nurse's child is taken from the mother's breast it becomes a prey to all the dangers that threaten the artificially fed babe; and, furthermore, that it runs additional risk from the fact that it often goes from the mother's care to that of an untrustworthy care-taker. As a matter of fact, the mortality among the children of wet-nurses is very great. This is well illustrated by the fact that during the siege of Paris the death rate among the infants showed a decided falling off in those districts from which the Parisian ladies usually obtained their wet-nurses, owing to the fact that during this time the mothers remained with their children. Again let me suggest the advisability of taking the child with the mother, a plan which for reasons given above may often be of direct benefit to the foster-child.

The nurse herself is often directly injured by being deprived of her milk through thoughtless treatment or by being regardlessly dismissed in a short time. So that whereas she might have been in the position to support herself and her child for months, her pay as nurse enabling her to hire a proper care-taker for her child, they are now, both mother and child, without means of support and are liable to suffer want. The child as usual suffers most, as it is young and has less power of resistance.

PATHOLOGY OF THE NURSING WOMAN

General Diseases.—Only such disturbances will be considered here as are peculiar to the healthy nursing woman. The first thing to be considered is constipation, that ever-present complaint of the nursing woman. I cannot but attribute the presence of this trouble, to a great extent, to the senseless regime which is oftentimes prescribed by the physicians themselves. The favorite insipid unappetizing diet of the nursing woman which produces but little peristaltic action; the perfectly incomprehensible prohibition of the use of fruit, together with a decrease in general activity, may be considered the etiological factors. Indeed, the use of large quantities of cow's milk, expedient as a matter of diet, often causes disagreeable constipation.

Therapy.—The diet should be regulated. Food rich in cellulose should be given (fruit, vegetables, lentils, huckleberries), and proper exercise should be ordered. If a laxative is necessary, castor oil should be avoided, since this, like other fat products, is taken up in the produc-

tion of the milk. Thus far no particular bad effects have been noted from its use, but it always seems better to use some of the many other evacuants, preferably a mineral salt.

Diseases of the Breast.—The very painful nodes which are the result of stasis border upon a pathological condition. The treatment, a complete emptying of the breast, is suggested by the etiology. Binding helps to relieve the condition.

Far more painful are the fissures, which are usually very much dreaded; they are as a general rule circularly arranged and are not always easily seen, but the great pain on nursing makes the diagnosis certain. The bad effects of these are felt in various ways. At first, the act of nursing is accompanied by great distress, but later, and this is the most important, the glands become infected by way of the fissures. The treatment must be first directed against the harmful effects of nursing. For this, particularly on account of the very severe pain, the child is allowed to nurse with a nipple shield, which does not always satisfy it. Where the distress is very great it may become necessary to put the child only to the sound breast and completely to empty the diseased one manually. The healing process is assisted by applications of nitrate of silver of six to ten per cent. solution, or the use of a salve composed of:

R Argen. nitr. 1.0. gr. xv
 Bals. peruv. 10.0. ℥ iiss
 Ung. paraffin. ad 100. q.s. ℥ iiii

Or tannin-alcohol, which is composed of:

Acid. tannic. 2-5. gr. xxx-lxxv
 Glycerin. 20.0. ℥ v
 Spir. rectificatiss. ad 100.0. q.s. ℥ iiii

M. Sig.—Apply frequently.

The French particularly recommend orthoform. A dusting powder or a ten per cent. alcoholic solution is used, rubbing it in for half an hour and then carefully washing it off.

Mastitis.—Inflammations of the lactating glands are often produced by bacteria, especially the staphylococci or the streptococci, the point of entrance being as a rule the fissures or cracks of the nipples. Accompanying an increase of temperature, the breast becomes painfully hardened, in spite of the attempt to empty it. Later the infiltration becomes increased, the skin begins to redden, and soon the fluctuation shows that purulent disintegration has begun. If this is not opened multiple perforations often occur spontaneously. In neglected cases, the whole organs becomes infiltrated and inflamed, pus being discharged from numerous openings.

Therapy.—In inflammations of the breast, prophylaxis is of the greatest significance. Where there are no points of entrance for bacteria there can be no inflammation. The slightest crevices in the nipple, therefore, to say nothing of the fissures, should be treated with the

greatest care. If a proper technic is carried out infections will surely be avoided.

If the condition is already present we should attempt to secure restitution by hydropathic applications and bandaging. Frequently this regime will accomplish the result. But when abscess formation has already begun, incisions should be made, and care should be taken to make these radially in order not to wound unnecessarily the numerous milk-ducts. Drainage tubes or strips of iodoform gauze should be placed in the incisions. Good results have also recently been obtained by the Bier suction apparatus.

Mastitis is Not Necessarily a Contraindication to Nursing.—So long as there are no pus corpuscles found in the milk, the child may be allowed to nurse. In other cases, where the pain from stasis in the inflammatory tissue is especially severe, it will be necessary to empty the breasts manually as far as possible, regardless of the quality of the milk.

On the healthy breast, nursing must, of course, be continued. It often happens that the productive power is so greatly increased that it is sufficient for the nourishment of the child.

METABOLISM AND NUTRITION DURING THE FIRST YEAR OF LIFE

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JUSTIFICATION is hardly required in a text book of children's diseases for the devotion of considerable space to the presentation of physiological conditions. A thorough foundation in physiology is especially indicated in pediatrics. The great progress of the natural sciences and of medicine within the last decade has considerably advanced our knowledge of the life of the normal healthy human organism. Each step of this progress leads to new questions and opens up new problems. Nevertheless, we affirm with satisfaction that, thanks to the labor of many investigators during the past thirty years, numerous problems have found a sufficiently exact explanation, and this is especially true in the physiology of childhood. It must be acknowledged that some of these problems had been worked out much earlier. We need only recall the very important investigations of Quetelet concerning the growth of man.* But for a surprisingly long time knowledge on most important subjects—particularly the nutrition and metabolism of children—was sadly deficient.

Allix, a Frenchman, contributed the first work on the physiology of infancy. The first observations on the quantity and character of the food were recorded by Forster, a pupil of Voit. But Vierordt, of Germany, is the real founder of the physiology of childhood. He published in 1877 for the first time, in Gerhardt's *Handbuch der Kinderkrankheiten*, a physiology comprising the entire period of childhood. To him we owe the very important knowledge of the surface of the body at different periods of life; and he was the first to try the determination of a metabolism equilibrium in the child and to calculate the absolute and relative expenditure of heat. For the latter, he found 130 calories per kilogram in an infant five months of age, and 91 calories in a child of a year and a half. These figures are not far removed from the real values. Lavoisier calculated the intake and expenditure of heat in the adult, and he regarded the production of animal heat as due to the formation of carbonic acid and water in the body. Boussaignault, Liebig, Dumas, and others tried to determine the heat produced from the difference

*Quetelet sur l'homme, Paris, 1835.

between the amount of carbon and hydrogen introduced into the body and the amount eliminated in urine and feces within 24 hours. They calculated that the adult requires 2400 calories per diem, a figure which approaches the values which Despretz previously obtained on the basis of direct calorimetric measurements. A complete understanding of the transmutation of energy could not be obtained before the discovery of the law of the conservation of energy (R. Maier). Camerer (the elder), Heubner, and Rubner deserve the credit of introducing the use of this law in the physiology of childhood. Despite the approximate correctness of his calculations, the explanation which Vierordt deduced from his results is not quite right, as we will show later on. We have to mention here the numerous observations and investigations of Camerer (the elder) concerning the metabolism from the time of birth to the end of the time of growth, and in regard to the growth in length and weight. With Söldner he made investigations into the chemical composition of human milk. The contributions of Rubner concerning the balance of energy, and the exact metabolism experiments which he conducted in conjunction with Heubner and other investigators, were of great importance. Valuable observations in regard to nutrition and metabolism were reported from obstetrical departments, while the numerous publications of former times from children's hospitals treated more of pathological conditions.

The reason which Camerer (the elder) gave in 1881 to explain the unsatisfactory result of artificial infant feeding shows how insignificant our knowledge was only a short while ago in regard to one of the most important points in practice. He deemed it necessary, in order to establish artificial feeding on any other than a rather crude empirical basis, to obtain clear information as to the infant's physiological requirement of water, proteids, fats, sugar, and salts, and as to the utilization of a normal food by the healthy infant. The problems of artificial feeding could be formulated in a really scientific manner, and rational methods of artificial feeding be instituted, only when such examinations had been made on a certain number of infants. The line of investigation has now been carried out in its essential points, and we are indebted to numerous researches of practicing physicians and patient investigators. Among the number of German authors who have contributed to the accomplishment of this task, the names of Biedert, Czerny, Escherich, Keller, Pfaundler, and Schlossmann should be mentioned.

At the present time we have at our disposal a mass of information with regard to the physiology of nutrition, which is of great value for the management of the infant's metabolism in health and disease. There are several reasons why this information has not been taken sufficient notice of by physicians and hygienists, and why its significance is not duly recognized. One reason is that it is relatively so recent in date.

Another and more potent reason is that a great number of students of medicine and many physicians have not sufficient opportunity to apply themselves to pediatrics, and particularly to the physiology of childhood, during the period of their professional education. The new medical examinations law in Germany requires proof from the candidate that he attended a children's hospital or dispensary regularly for half a year. According to the report of Heubner, in one-half of all the Prussian universities neither exists, while the percentage of universities in which a thorough study of the physiology and pathology of the infant is made is still lower. One of the circumstances which may explain this surprising neglect of such an important branch of medicine can be found in the fact that even to-day many physicians, and even prominent clinicians, deny to pediatrics the right to exist as a specialty. Certainly, there is no sharp line of demarcation in the gradual transition from the child to the adult, which would permit a clean separation of the two subjects, and later childhood may be claimed with a certain degree of justification to belong to the sphere of internal medicine. But this cannot be said of the period of infancy and of early childhood.

A glance at the exterior build of a child shows that it can by no means be regarded as a small edition of an adult, and a closer examination of its anatomical and physiological conditions demonstrates a number of peculiarities which cannot be explained by a simple comparison with the adult, but require a special study. The period of childhood is not an indivisible unit, and with Vierordt we distinguish the following subdivisions:

A. Childhood:

1. Infancy:

- (a) Period of the newborn (first week of life).
- (b) Period of suckling (until the eighth month of life).
- (c) Period of the first dentition (until the fifth year of life).

2. Childhood, infancy to puberty.

B. Puberty.

Each of these periods has its physiological peculiarities, which in the period of the newborn and of the suckling are chiefly those of metabolism.

It is necessary at first to obtain information about the chemical composition of the infant's body, in order to understand the conditions of the metabolism at these periods of life.

On account of the great difficulties in procuring suitable material for such investigations,—the cadavers must be of normal individuals, and must not have suffered essential changes of the composition in consequence of the causes of death,—thus far we have only complete analysis of the newborn. It would be of the greatest advantage to know the composition of older children, so as to become acquainted with the

changes brought about in the composition of the child by growth. Furthermore the nature of growth in its different periods could be learned, which otherwise can only be accomplished with difficulty and uncertainty. For instance, if the quantity and chemical quality of the intake and of the excretion of a child, including the gaseous metabolism, are determined and compared, the difference will show how much of the different substances (proteid, fat, water, ash) have remained in the body. All determinations of these differences suffer from the fact that all the errors of the experimentation accumulate themselves on the values for the differences, and if the differences are small, the result becomes very uncertain (see p. 367). At the time of the most energetic absolute and relative growth, which is toward the end of the first month of life, the daily gain in weight amounts to only about 30 Gm. (1 ounce). This would mean about 4 per cent. of the total intake or excretion, and later on only two per cent. are accounted for in the gain of weight. The analyses of the newborn, however, permit us to draw conclusions in regard to the substances which combine to make up the gain, since we may be justified in assuming that the composition of the bodies of children varying in age a few weeks or months does not differ materially from that of the newborn. This assumption is strengthened by some analyses of pathological older children (Sommerfeld, Weigert, Steinitz).

The first, though incomplete, data were furnished by Bischof and Fehling. Within recent years the writer and Söldner have analyzed the bodies of six normal newborn infants, according to a method proposed by Camerer (the elder). Each body was frozen and then ground up. The whole material was extracted with alcohol and ether, and the resulting substance (poor in fat and water) was pulverized. The individual analyses agreed very well with each other, and the following average values for one infant were obtained:

TABLE 1.

	Body weight. ¹	Water.	Solids.	Fat.	Ash.	Proteid.	Extractives. ²
Absolute values.....	2820	2026	795	348	75	330	42
100 Gm. body contain.....		71.8	28.2	12.3	2.7	11.7	1.5
100 Gm. dry residue contain.....				43.8	9.4	41.5	5.3

The analyses of chemical elements gave the following average values:

TABLE 2.

	C.	H.	N.	O.
Absolute values.....	449.6	67.15	55.8	147.45
100 Gm. body contain.....	15.9	2.38	1.98	5.36
100 Gm. dry residue contain.....	56.5	8.4	7.0	18.7

¹ The low birth weight is due to the reason that the weight of meconium, urine in the bladder, stump of the cord, and vernix is already subtracted, and that all the infants were born by small mothers of the working class.

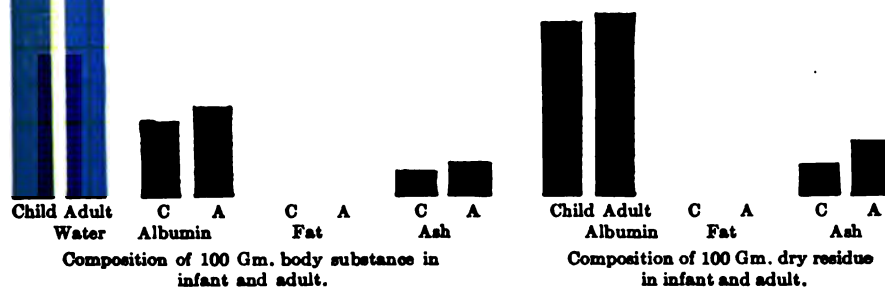
² The extractives are calculated from the difference.

In the two figures following the values which Volkmann found for the adult are added for comparison:

FIG. 50



FIG. 51.



The body of the newborn is relatively richer in water and fat (the latter constitutes nearly one-half of the dry residue) but poorer in nitrogenous material, and particularly in ash, than is that of the adult. The relative small amount of muscle and bone in the newborn will explain this fact. According to Vierordt, the muscles of the newborn represent 23 per cent. of the total body weight, while those of the adult represent 43 per cent. The skeleton represents in both cases 16 per cent., but the composition of the latter differs very considerably in the infant and in the adult. Neither the sex nor the absolute body weight of the infant has any influence on their composition; nevertheless, it appears probable that heavy newborn infants of well-to-do mothers are richer in fat.

Seeing how much the composition of the newborn differs from that of the adult, a comparison with that of the foetus at different ages is of great interest. On the basis of his investigations, Fehling gives the following data:

TABLE 3.
The foetus contains, in per cent. of the total amount:

Age.	Water.	Ash.	Fat.	Proteid.
6 weeks	97.54	0.001		
4 months	91.79	0.98	0.57	4.87
1st half of 5th month	90.70	1.4	0.48	5.9
2nd half of 5th month	90.7	1.43	0.54	6.0
6 months	89.2	1.94	0.72	6.67
7 months	82.6	2.94	3.47	11.8
8 months	82.9	2.82	2.44	10.4
9 months	74.7	3.3	8.7	12.6

The most striking feature is the large proportion of water; the younger the foetus, the larger the percentage.

The conditions after birth are similar. As previously shown, the adult is relatively poorer in water and richer in ash than is the infant, and it may be assumed that in the aged the body contains more ash and less water than does that of the adult, although there are no exact investigations extant. From the early foetal period until old age the relative water content of the body decreases, while its ash content increases. This process of drying out helps to explain why the metabolism is more active in the early foetal period than later, since the higher the percentage of water, the easier are the processes of metabolism carried on.

The investigations of Giacoso, de Lange, Michel, Söldner, and Hugounenq teach us the composition of the ash of the foetus and of the newborn.

TABLE 4.
In 100 parts of the ash of the foetus Hugounenq finds:

	Period of pregnancy.				
	4-4½ months.	4½-5 months.	5-5½ months.	6 months.	6½ months.
CO ₂		1.5	0.96	0.90	0.32
Cl.....	8.99	9.91	8.59	7.75	8.53
P.O ₅	37.74	32.33	34.36	34.94	35.39
SO ₃	1.46	1.27	1.80	1.78	1.46
CaO.....	32.60	38.21	32.50	34.60	34.13
MgO.....	1.47		1.58		1.17
K O.....	9.12	1.21	8.28	7.21	8.45
Na O.....	12.23	13.75	12.62	10.62	10.95
Fe.O ₃	0.43	0.33	0.40	0.39	0.38

TABLE 5
In 100 parts of the ash of a newborn infant Söldner finds:

	K ₂ O	Na ₂ O	CaO	MgO	M ₂ O ₃	Fe ₂ O ₃	Mn ₂ O ₄	S ₂ O ₃	SO ₃	Cl	SiO ₂	CO ₂
Newborn.....	7.06	7.67	38.08	1.43	0.11	0.83	0.03	37.66	2.02	6.61	0.06	0.53
Human milk.....	32.4	13.1	13.9	1.9		0.07		11.4	3.3	21.7		

The knowledge of the ash content of the newborn enables us to test the validity of Bunge's theory. He found that in numerous families of mammals, the percentage composition of the ash of the newborn and of the mother's milk was about the same, with the exception of iron, while the ash of the blood differed very much, as the following table shows:

TABLE 6.

	K ₂ O	Na ₂ O	CaO	MgO	Fe ₂ O ₃	P ₂ O ₅	Cl
Suckling puppy.....	8.5	8.2	35.8	1.6	0.34	39.8	7.3
Milk of the dog.....	10.7	6.1	34.4	1.5	0.14	37.5	12.4
Blood of the dog.....	3.1	45.5	0.9	0.4	9.4	13.3	35.6

From these figures, Bunge concluded that the epithelial cells of the mammary glands collect selectively all the inorganic materials from the blood in exactly those proportions which the offspring requires in

order to grow and to become equal to the organism of its parents, and that therefore the proportions of the inorganic substances in the total organism of the suckling are nearly identical with those in the mother's milk. He transferred his conclusions to the human species.

Söldner's analyses of the ash of the newborn and of human milk are given in Table 5, and a comparison of the figures shows that this hypothesis is not correct. In a more recent paper Bunge corrected his opinion. He claims that the ash of the newborn of different species of mammals seems to have nearly the same composition. But the slower the suckling grows, the more the ash of the milk differs from that of the offspring. The milk ash becomes richer in alkalies combined with chlorine and relatively poorer in phosphates and sodium chloride. Bunge explains this on teleological grounds. He claims that the ash of the milk has to serve a double purpose. It has to furnish material for the construction of the tissues, and also for the excretions, particularly the urine. The more rapid the growth of the suckling, the more the first purpose preponderates; the slower the growth, the more the second. The percentage figures cited by Bunge seem to bear out this opinion.

The proportion of the individual constituents of the ash of the milk is one of the numerous factors which determine these processes of metabolism. But in reality the absolute amounts of the ash introduced must be taken into consideration (see Table 13, p. 377). A comparison of the ash constituents of the milk and of the body is not sufficient in the present state of our knowledge to explain satisfactorily the conditions of the inorganic metabolism. It may readily be that the combination of the metals and of the other elements with organic molecules, and their presence in the form of inorganic compounds or ions in the milk differs very much from their arrangement in the body substance.

The digestive organs of the infant differ in many points from those of the adult. The peculiarities that are of most practical importance will be briefly considered.

The development of the salivary glands begins during the second month of the foetal period. This secretion has the power to decompose starch in the first days of life. The quantity of the secretion is small because chewing, the most potent physiological stimulus, is wanting. Therefore only little starch can be decomposed but the secretion becomes more abundant when the teeth make their appearance with the gradual transition from liquid to solid food. The reaction in the carefully cleansed cavity of the mouth is neutral or weakly alkaline. The acid reaction which is frequently observed, is due to decomposed particles of food.

The characteristic features of the infant's stomach are (1) its form and position, (2) its small capacity, and (3) its insignificant secretions.

According to Fleischmann, the cardia is fixed to the left of the tenth dorsal vertebra, the pylorus is situated 2 to 3.5 cm. lower down in a prolongation of the middle sternal line, but rarely to the right of this line, and occasionally even somewhat to the left. When the stomach is empty, the pylorus forms its lowest point. In most cases the fundus is distinctly formed in the newborn, but it remains relatively flat for some time; and this, in conjunction with the position of the stomach, explains the frequency of vomiting and regurgitation in infants.

Pfaundler made a thorough study of the capacity of the stomach. From the first to the twelfth month he found the following values: 90, 100, 110, 125, 140, 160, 180, 200, 225, 250, 275, 290 c.c., (3, 3½, 4, 4½, 5½, 6, 6½, 7½, 8½, 9, 9½ ounces) respectively.

These figures can serve only as approximate guides for the size of a single meal, because considerable individual variations of the stomach capacity exist, and a part of the stomach contents passes into the intestine during drinking. The emptying of the stomach is dependent on the amount and the quality of the food. The stomach of the breast-fed infant is found empty after an hour and a half if small meals are given. If large meals are given, it takes two hours before the stomach is empty. The bottle-fed baby requires two and one-half to three hours to dispose of a large meal.

The secretory function of the stomach is already developed in the newborn. The mucous membrane of the embryo's stomach has an alkaline reaction (Toldt), but in the still-born the reaction is neutral or acid. It is always acid after ingestion of milk, and this is due to the secretion of hydrochloric acid. At first the acid combines with the proteids and salts of the milk, and appears as free hydrochloric acid only after their saturation. In healthy breast-fed infants it is found about two hours after the meal in a concentration of about 0.1 per cent. In bottle-fed babies, free hydrochloric acid occurs much later or not at all, because the power of cow's milk to combine with the acid is much greater. Therefore, the stomach contents of the bottle-fed infant are less antiseptic than are those of the breast-fed. Beside hydrochloric acid, the presence of lactic acid can be demonstrated in the infant's stomach contents. But nothing is known concerning its origin or its action.

The stomach of the infant contains rennin and pepsin the latter occurring in the embryo. The rennin causes the coagulation of the milk casein in the stomach, the coagulum being dissolved by the hydrochloric acid. The casein of cow's milk coagulates much more completely than does that of human milk, and only a part of it is dissolved by the acid (Biedert). Mare's milk is the only milk resembling human milk in this regard, and this is poor in proteids (Langgaard).

Nothing definite is known about the action of pepsin in the infant's stomach. The stomach contents of the infant can be shown to contain

peptone, but this could be due to the action of rennin or to bacterial decomposition.

The first scientific investigations of the intestinal tract of the infant were conducted by Schwan. He discovered the interesting fact that the length of the intestines as compared with the length of the body is considerably greater in the infant than in the adult. This corresponds to the relatively greater requirement of food. The proportion of the length of the intestines to that of the body is in the infant about 6 or 8 to 1, in the adult 5 to 1. The absolute length of the intestines in the newborn is subject to wide individual variations, reaching about from 200 to 400 cm. (6½ to 12 feet). Gundobin found the length and height of the folds of the small intestine in the newborn less, and the number of the villi greater, than in the adult. The muscles of the intestines are relatively poorly developed in the newborn. The development of the glandular tissue is the same as in the adult, while the lymphatic apparatus (solitary follicles and Peyer's patches) is relatively more developed. Orban and Weinland have lately studied the intestinal secretions. According to these authors, the secretion of the small intestine in the newborn contains a ferment, lactase, which is capable of decomposing lactose. Moro and Jakubowitsch, in contradiction to Korowin and Zweifel, affirmed that the pancreas secretion of the newborn possesses a diastatic activity, which is small when compared with the lipolytic and proteolytic activity.

The liver, the second large abdominal gland, is of an extraordinary size in both foetus and infant. According to Harley the weight of the liver in relation to the body weight is in the newborn as 1 to 18; in infancy as 1 to 20; at puberty as 1 to 30; in the adult as 1 to 35; in middle age as 1 to 40; in old age as 1 to 50.

The investigations of Zweifel show that the secretion of bile begins in the third month of foetal life. In the fifth month the formation of glycogen in the liver can be demonstrated.

GENERAL CONSIDERATIONS CONCERNING THE METABOLISM OF THE INFANT

Changes in the condition of the body cannot be observed directly, but can be calculated indirectly from three factors of metabolism. We have, however, to make certain assumptions with regard to the carbon metabolism. The three factors are, the food, the excretion of urine and feces, and the gas metabolism (intake of oxygen, excretion of carbon dioxide and water vapor). The change in the body of a healthy breast-fed baby (as shown in the gain of weight) rarely amounts to more than 40 Gm. (1½ ounce) in 24 hours. This value, like all values deduced from a comparison of differences, is rather uncertain and inexact. In the tenth week of life the figures for the ingestion and excretion amount to

not less than 800 Gm. (25 ounces), so that in the calculation of the metabolism all the errors of the metabolism accumulate themselves on this small value. The gaseous metabolism can only be determined by means of the respiratory apparatus of Pettenkofer. In the adult this arrangement is very likely to alter the normal conditions of life and of the experiment, and it always does so in the case of the infant.

The infant, whose conditions of life are changed by being put in the apparatus must be so arranged as to permit a separate collection of urine and feces without loss. The child becomes very restless and fretful, and this exercises an influence on the metabolism. The gaseous metabolism is thus increased with an increased excretion of water vapor, which is still further encouraged by the abnormally high temperature of the air in the apparatus. Consequently, the urine is more scanty and more concentrated than normal. Furthermore, the infant cannot remain in the apparatus more than twenty hours in twenty-four, as it must be removed at least five times to take food. This time is utilized to empty the vessels containing the urine and feces, to change clothes, bedding, etc. Thus for four very important hours, the gaseous metabolism could not be determined.

The successful and exact accomplishment of the experiment requires from four to six days; it is very difficult, and beside complicated pieces of apparatus numerous well trained investigators have to be employed. In the course of the last few years, Heubner and Rubner, with numerous co-workers, have conducted several of these metabolism experiments on infants of different ages. Although the results were very interesting, they were unable to obtain satisfactory information in regard to the conditions prevailing in the normal breast-fed infant, and particularly concerning its gain. This is due to several reasons. There was a constant error in the form of an excessive gaseous metabolism, and the small number of infants examined, with the short duration of the experiments, permitted too great a range of individual peculiarities.

There is another method for determining the values of metabolism, which can be designated as the *statistical method*. This is of particular interest to the practicing physician, because he is able to apply it himself and it enables him to participate in the investigation of the subject. The results thus far obtained in this field have mostly been furnished by the work of the practitioner. For instance, during the total period of lactation the average quantity of the milk and its value in nutrient material were determined, as well as the average amount and composition of urine, and feces, and the gain. This was done without altering the conditions of the baby's life. This method leaves the gaseous metabolism unknown, but it can be calculated with a high degree of certainty. The values involved are 100 Gm. (25 drams) or more, and are only a little influenced by the very insignificant uncertainties inherent to the determination of the three other factors of the metabolism.

The statistical method gives very reliable average values for the factors of metabolism in the normal breast-fed infant. A comparison of these average values with the results of the individual experiments makes it possible to recognize the influence of constant errors or of individual peculiarities, as well as the absence of such disturbances. For the reasons given above, such irregularities in the infant will hardly be avoided and therefore the results of the calculation will rarely coincide with those of the direct observation. Rubner observed the metabolism of two boys ten and eleven years of age in the respiration apparatus, and compared the results with those obtained according to the statistical method by Camerer (the elder) for boys of this age and found an unexpected similarity.

Whether or not such average values can be of practical use depends on how much the values vary with regard to individual infants and to single days or weeks of life. A complete agreement of the individual case with the famous "ideal" average values hardly ever occurs in the infant, either in observation or in the calculation of probabilities.

The great majority of cases do not deviate widely from the average values. When constant influences are at work, such as the restlessness produced in the infant in the direct metabolism experiment, the deviations from the normal may be such that they must be taken into consideration. Careful observers have always noted these conditions, inasmuch as certain cases have been regarded as comparable while others were excluded from the comparison. For instance, an infant perspiring profusely, for some reason or other cannot be expected to void the same amount of urine as another infant not otherwise differing from the first under normal conditions of life. Or if an infant ten weeks old receives only 600 Gm. (21 ounces) of breast-milk instead of the normal amount of 800 Gm. (28 ounces), and its weight, owing to general debility, is only 4.4 kg. (10 lbs.) instead of the normal 5 kg. (11 lbs.), these two circumstances have a marked influence on the functions of its metabolism, which must be taken into consideration in comparing the metabolism with that of a normal infant. If a comparison is desired, the amount of food required in the given case would approach the amount for an infant of seven weeks rather than that for one of ten weeks, etc.

By way of explanation, two complete balances of metabolism and energy of infants in the tenth week of life may be cited. The average values of Infant 1 were obtained by the statistical method, the figures for Infant 2 are taken from the first experiment of Heubner and Rubner, which lasted nine whole days—24-hour periods. The amount of food in Case 2 was insufficient, and the infant showed a tendency to diarrhoea.

The following values were used for the calculation in Case 1, living under normal conditions: Weight 5 kg., daily gain 25 Gm., with 3.7 Gm. proteid, containing ash and 3.3 Gm. fat. The so-called extractive

substances, like glycogen, lecithin, etc., cannot be considered in such calculations, and their amount is only small. The daily food is taken to be 800 Gm. breast-milk, with 7.6 Gm. proteid, 28 Gm. fat, and 56 Gm. lactose. All other figures can be seen in the table. Infant 2 weighed at the beginning of the experiment 5220 Gm., and at the end 5250 Gm., the average was taken as 5230 Gm. It drank daily 613 Gm. mother's milk, with 6.15 Gm. proteid, 17.1 Gm. fat, and 43.5 Gm. lactose. These figures and those of the following table were obtained by special analyses. The mother of the infant thought that the flesh of the baby had become more flabby than usual and the authors indicate that this observation was correct. On account of the insufficient nourishment, the calculation of the balance of energy showed a slight deficit.

TABLE 7.

INFANT 1.

	(a) Food: milk 800 Gm.	(b) Gain: 25 Gm.	(c) Remain for excretions: 775 Gm.	Excretions.		(f) Remain for gaseous excre- tion: 235 Gm.
				(d) Urine: 520 Gm.	(e) Feces: 20 Gm.	
Water	708	18	690	517	16.2	156.8
Ash	1.4	0.7	0.7	0.5	0.2	0
Organic substance.....	90.6	6.3	84.3	2.5	3.6	78.2
Organic substance composed of	N	1.3	0.5	0.6	0.2	0
	C	45.4	3.9	41.5	2.2	38.4
	H	6.8	0.6	6.2	0.3	5.7
	O	37.1	1.3	35.8	0.9	34.1

INFANT 2.

	Intake : 709.2 Gm.		Excretion : 704.2 Gm.					
	Milk : 613.	Oxygen through lungs : 96.2.	Urine and sweat : 324.7.	Feces : 38.2.	Gaseous excretion : 341.3.		Sum of the excre- tions.	Balance for the gain— 5 Gm.
					CO ₂ : 116.9.	H O : 224.4.		
Water	524.7		322.0	35.0		181.4	538.4	4.3
Ash	1.3		0.8	0.3			1.1	0.2
Organic substance.....	69.0	96.2	1.9	2.9	116.9	43.0	164.7	0.5
	165.2							
Organic substance is composed of {	N	1.0		0.5			0.7	0.3
	C	34.4		0.6	1.9	31.9	39.4	0
	H	5.2		0.2	0.2		4.8	0
	O	28.4	96.2	0.6	0.6	85.0	38.2	124.4
		124.6						

Explanation of Infant 1.—The 38.4 Gm. carbon of column (f) required for oxidation 102.5 Gm. oxygen; the 5.7 Gm. hydrogen required 45.6 Gm. oxygen; together 148.1 Gm. oxygen. From the ingested milk only 34.1 Gm. remained; therefore, 114 Gm. oxygen must be taken up through the respiration. The respiration excretes 140.9 Gm. carbon dioxide, and the skin and lungs excrete 208.1 Gm. water, 51.3 Gm. of

which are produced in the body through oxidation and 156.8 Gm. come from the ingested milk. The loss through the gaseous metabolism (the so-called insensible perspiration) is $114 - (140.9 + 208.1) = 235$ Gm.

Explanation of Infant 2.—From 31.9 Gm. carbon and 85.0 Gm. oxygen and from 4.8 Gm. hydrogen and 38.2 Gm. oxygen, 116.9 Gm. carbon dioxide and 43.0 Gm. water are produced in the body through oxidation. Since carbon and hydrogen were derived from the organic substance, the carbon dioxide, the newly formed water, and the oxygen of the respiration had to be registered on the line of the organic substance. The 224.4 Gm. water eliminated through the skin and lungs are derived from 43.0 Gm. produced in the body and 181.4 Gm. of the milk. The insensible perspiration is $96.2 - (116.9 + 224.4) = 245$ Gm.

Balance of Energy for Infant 1.

Intake of food calories (calories in total)	520
Subtracted for feces, skin excretion, etc	40
Subtracted for gain (utilized calories)	50
Expended calories (utilized calories)	90
	430

For 1 kg. body weight there are 104 total calories; and 86 utilized calories were expended.

Balance of Energy for Infant 2:

Intake of total calories	370
Subtract for feces, loss through skin, etc.	30
Difference	340
Added from the excess of oxidized body fat (3 Gm.) above the gain in organ substance (8 Gm.)	10
Calories expended (utilized calories)	350

For 1 kg. body weight there are 72 total calories, while 67 utilized calories were expended.

All the calorimetric values were obtained experimentally.

Balance 2 is more difficult to explain than is Balance 1 on account of the existing anomalies, the insufficient nourishment, and the excessive elimination of water vapor. Much crying caused a great loss of water vapor through the lungs, and the abnormally high temperature in the apparatus (about $25^{\circ}\text{C}.$; $77^{\circ}\text{F}.$) caused the loss through the skin. In spite of the small amount of milk taken in Case 2, the excretions through feces and the gaseous excretions do not differ very materially from those of Case 1 (20 Gm. as against 38 Gm., and 349 Gm. as against 341 Gm.). A considerable difference in regard to the excretion of urine is noted (520 Gm. as against 325 Gm.).

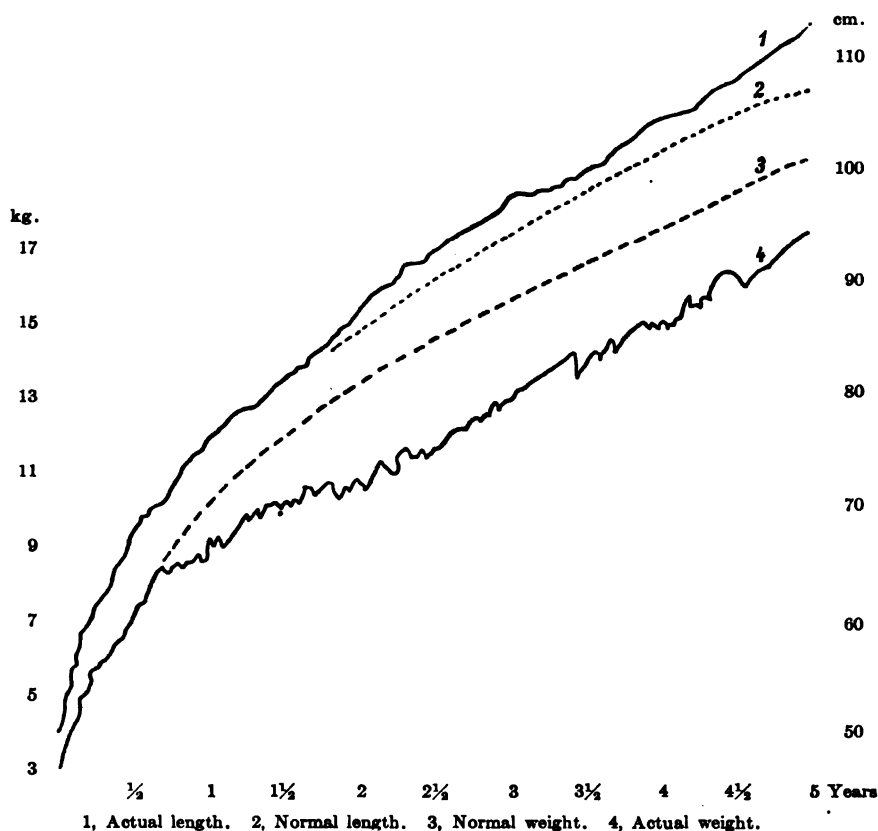
The so-called respiratory quotient* was the same in both cases, in Infant 2, $\frac{85}{96.2} = 0.88$ (for the figures of the fraction see line for O in Table 7); in Infant 1, $\frac{102.5}{114} = 0.90$ (see explanation given above).

* The respiratory quotient (oxygen of the carbon dioxide divided by the oxygen derived from the air) equals 1 when carbohydrates are oxidized; equals 0.78 when proteid is oxidized; equals 0.72 when body substance is oxidized.

From this the conclusion is drawn, that a relatively large amount of carbohydrates (lactose in this case) was oxidized in both cases. Infant 2, might have registered a fair gain in spite of the insufficient amount of food, if it had been less restless and had cried less. Under the prevailing conditions the observers calculated a slight gain in body substance with a loss of some body fat. Such peculiar conditions of growth as these are occasionally observed in children who do not receive a sufficient amount of food.

Fig. 52 illustrates such a case. This child suffered with a geo-

FIG. 52.



graphical tongue from the ninth month to the second half of the fifth year and therefore did not eat sufficiently. Attacks of sickness can be recognized by the occasional losses in body weight. This boy received the breast for three months and the bottle up to nine months, with good result, before the affection of the mouth influenced his gain in weight.

The growth in length (in the skeleton and partly in the muscle) even exceeds the normal from 1½ years on, while the gain in weight remains far below the normal and does not begin to rise until late. There

is every reason for assuming that the chemical composition of the body is normal, with exception of the fat. The gain in proteid and ash therefore was about normal while the gain in fat was abnormally low.

It would be possible to give the balances of metabolism obtained by the statistical method for each period of infancy (with the exception of the first week of life, which will be considered separately) for the breast-fed infant as well as for the one artificially fed. But the one example will suffice. On this basis, the significance of the total amount of food and of the individual constituents of the food may be learned, with special reference to the life of the infant. A few more data concerning the gaseous metabolism at different periods may be cited here.

TABLE 8.
The average insensible perspiration amounts in grams, to:

Period of life.	In 24 hours.	In 1 hour.	Per kg. per hour.
End of 1st week.....	110	4.5	1.3
End of 4th week.....	130	5.4	1.3
End of 10th week.....	180	7.5	1.6
End of 15th week.....	220	9.2	1.6
End of 25th week.....	290	12.1	1.7
Adult.....	1200	50.0	0.8

The variation in the amount of insensible perspiration is considerable; for instance, a young infant eliminates 2 to 3 Gm. (30–45 grains) if resting well, but if very restless 10 to 15 Gm. (150–225 grains) per hour.

Formerly, an excessive influence on the processes of the metabolism was attributed to the growth of the infant, and it is generally thought that infants and children require relatively a large amount of nourishment, "because they grow so much." But this is not well founded. The daily gain of the 10-week old infant of Table 7 was only 25 Gm. (6 drams), equal to 0.5 per cent. of its body weight; while at the end of the first month, which is the time of the most active absolute and relative growth, the average daily gain is 30 Gm. (8 drams) or just about 1 per cent. of the body weight.

At the present day, in the light of the theory of energy, it is not conceivable how a gain of only 1 per cent. or 0.5 per cent. should exercise a leading influence on the required amount of food. Table 7 (Infant 1) demonstrates that not less than 85 per cent. of the ingested organic substance is oxidized and eliminated through the skin and lungs in the form of carbon dioxide and water. This means that more than 80 per cent. of the introduced energy is thus eliminated, while only about 9 per cent. remains in the body to constitute the gain. In the balance of Infant 2 a gain in body substance took place, although the food was insufficient and the balance of energy was negative. Such observations and considerations enforce the conclusion, that the relatively great food requirement must be due to peculiarities of the infant's body. The growth must be ascribed especially to those peculiar forces which, after

being set in motion by fertilization, cause the segmentation of the ovum and the whole further development of the embryo. These forces attract substances to the growing cells of the body with such power that the growth can proceed even when at the same time body fat is decomposed. It was necessary to find out these peculiarities of the child's body. According to a known law of stereometry, the smaller of two bodies of similar form has relatively the larger surface,—that is, in relation to its cubic content, or to its weight if the two bodies are composed of the same substance. In the latter case it may be stated that the surface of 1 kg. of the small body is twice or three times as large as that of 1 kg. of the larger body. The consequence is that 1 kg. of the small body in the same time and under the same conditions can give off (or take in) twice or three times as much heat as 1 kg. of the large body. This can be readily illustrated with an example taken from daily experience: Suppose a hot water heater contains a kg. water and has a smooth surface of b square decimetres. If the surface is increased to $3b$ square decimetre by channeling, the amount of heat given off under otherwise equal conditions will be three times as great as that given off from the smooth surface.

Rubner justly remarks that it was not permissible to transfer the stereometrical standards of inanimate objects to the smaller animals, since the possibility of compensation exists (thicker fur, deposit of fat, or change in circulation). Experiments had to decide this question, but now we know that these modes of compensation can only furnish a moderate protection. The infant possesses only a slight power of compensation, as compared with the adult and the relative size of its body surface. The importance of the body surface in the determination of the balance of energy can be understood from the fact that the human being in a state of quiescence eliminates through the skin, in the form of heat and water vapor not less than 85 per cent. of the energy to be given off. Vierordt took steps to elucidate these peculiarities of the infant's body. He succeeded in finding a method which permits the calculation of the surface (of man and animals) from the weight. The direct measurement of the surface is a very difficult procedure. His assistant, Meeh, measured the surface of a number of people at different periods of life, and found that it can be approximately determined according to the formula $S = C^{\frac{2}{3}} \sqrt[3]{a}$, where S is the surface, C is a constant in man = 12.3,* and a is the body weight in grams. By the use of this formula, the table on the following page, showing relation between weight and surface in man was calculated.

An infant is seen to have a surface two to three times larger per

*Lissauer regards this figure as far too high for the infant, and substitutes $C=10.3$ (see W. Lissauer, *Jahrbuch für Kinderheilkunde*, 1903, vol. 58, p. 403).—TRANSLATOR.

kilogram than the adult. The food requirement of the adult is 35 calories per kilo body weight, while that of the infant is given as 105 to 110, or about three times as great, which facts should be brought into relation with each other. Vierordt studied in the calf the relations of gain, body substance already present, and food requirement, as the amount of milk used by the infant and its growth had not been deter-

TABLE 9.

	At birth.	10 weeks.	22 weeks.	1 year.	3 years.	Adult.
Weight in kg.....	3	5	7	10	15	70
Surface of body in sq. dcm.....	25	35	44	55	78	209
Surface per sq. dcm. to 1 kg.....	8.3	7	6.3	5.3	4.9	3

mined with sufficient exactness. The calf grows much more rapidly than does the infant and retains a much larger amount of food, namely, 60 to 70 per cent. of the proteid and fat. It is not surprising, therefore, that Vierordt adhered to the old opinion, that the processes of growth are the factors determining the necessary amount of food and energy in all growing organisms. He was the first to tabulate the relations between body surface and energy requirement of man for all periods of life.

Rubner's experiments on full grown dogs of different sizes completely cleared up the question. The amounts of carbon dioxide eliminated by these animals, which were observed under the same conditions, were equal when calculated in relation to the square metre, but were very unequal when calculated per kilogram of animal. Under certain conditions, the amount of carbon dioxide can be regarded as a measure for the energy expenditure of the body, and in the full grown animal the expended energy must be equal on an average to the introduced energy. Rubner concluded from his results that the relatively large requirement of food of small animals is dependent on the relative extent of their body surface, and he extended his views to the growing child.

An investigation on a full grown dwarf, conducted by Rubner and E. Voit, confirmed Rubner's theory. The weight of the dwarf was 6570 Gm., corresponding to that of a well nourished infant in the sixteenth week of life, and he took in about 630 calories in 24 hours, or 1500 to the square metre of body surface. This would be a little more than an infant of the given age does in spite of its daily gain of 24 Gm.

The size of the body surface is not the only factor determining the amount of food required. The gain in weight enters into consideration, although this factor is of much greater importance in rapidly growing animals than in the infant. Furthermore, the work of the digestion plays a rôle, as well as the powerful influence which the activity of the muscles exercises on the metabolism. The influence of hot or cold

can be nearly eliminated by means of clothing and dwellings. With the exception of the hours immediately after birth, the infant is more frequently exposed to overheating than to excessive cooling. Zuntz published investigations* which are of importance with regard to the influence of work on the expenditure of energy, and his results are here of interest. His data are not given in calories, but in kilogram-metres, 427 kilogram-metres being equal to 1 calorie.

TABLE 10.

	Light work : horizontal movement of 1 kg. animal on 1 m. level way; moderate velocity.	Heavy work : vertical movement, lift- ing 1 kg. animal to a height of 1 m.
Dog	0.50 kg.-m.	3.1 kg.-m.
Man	0.26 kg.-m.	3.1 kg.-m.
Animal	0.14 kg.-m.	2.9 kg.-m.

When light work was done the energy used (calculated from the introduction of oxygen) was nearly proportional to the body surface of the animals examined, the proportion being more exact in the resting animals. When the work was heavy, energy was used in proportion to the lifted weight, that is, to the performed labor. Such findings were to be expected from the statistics of the nutrition of human beings living under normal conditions. In the following table, the intake and expenditure of energy, calculated per square metre of body surface, varies more at the different periods of life than was to be expected from the results obtained by Rubner on dogs. But this is not surprising, and the deviations from the average values may be easily explained.

TABLE 11.

	End of first week of life.	10th week.	Child 9 years old.	Adoles- cent.	Man.	Old age.	
						Early.	Late.
Body weight in kg.	3	5	25	50	70	65	60
Body surface in sq. dm.	25	35	105	170	210	200	190
Food requirement for 24 hrs. in calories	210	500	1500	2200	2700	2200	1700
Calories for 1 sq. dm.	8	14	14	13	13	11	9
Calories for 1 kg. body weight.	70	100	60	44	40	34	30

Table 7 contains sufficient information about the significance of the individual constituents of the food for the metabolism of the infant. About 40 per cent. of the introduced nitrogen and just about 10 per cent. of the introduced carbon and hydrogen are retained; from this it may be calculated that about 40 per cent. of the introduced proteid and 12 per cent. of the introduced fat are used to form body substance, while all the rest, together with nearly all the lactose, is given off in urine, feces and (by far the greatest part) in gaseous form through the

* Ueber den Stoffverbrauch des Hundes bei Muskelarbeit, *Pflüger's Archiv*, vol. 36, p. 191. Zunts did not interpret his own results rightly. Camerer (the elder) gave the proper interpretation in the *Jahrbuch für Kinderheilkunde*, vol. 51, p. 51.

skin and lungs. Not less than 50 per cent. of the milk ash is retained, 36 per cent. enters the urine and 14 per cent. is excreted with the feces. The significance of the organic food constituents for the economy of the growing body is very strikingly brought out in the following table (values for 24 hours in grams):

TABLE 12.

	Proteid.	Mineral substances.	Fat.	Carbohydrates.	Water.	Water excreted per 1 kg. body weight.
1 kg. adult eats in Gm.	1.4	0.4	1.4	4.9	85	40
1 kg. infant, 10 weeks old, eats in Gm.	1.5	0.3	5.6	11.0	140	148

It becomes evident that proteids and mineral substances in the adult serve mainly to replace used up substance, while in the infant in addition to this they must help to make up the gain. Fat and carbohydrates serve mostly as sources of energy. Water is of importance as "Ersatzstoff"; beside this, the infant needs liquid food on account of the construction and properties of its digestive organs. The elimination of the water is of great importance in the infant's energy metabolism. The resting infant disposes of 60 per cent. of the expended energy by radiation of heat, and 35 per cent. is given off by evaporation of water through the skin and lungs. The corresponding figures in the adult are 73 per cent. and 22 per cent.

From this calculation it may be seen that the preservation of the proteid and ash content of the infant's and adult's body requires the introduction of 0.9 Gm. proteid for 100 Gm. proteid present in the body, and of 0.5 Gm. ash for 100 Gm. ash present. Older children perhaps require an introduction of 2 per cent. proteid to fulfil this purpose, the reasons for which need not be considered here.

The statistical method furnishes reliable values with regard to the introduction of the individual ash constituents. From Söldner's analyses the following values were calculated for a breast-fed infant on the eighth and the seventieth day of life, assuming the respective quantities of food in 24 hours to be 500 Gm. and 800 Gm. milk (values for 24 hours in milligrams).

TABLE 13.

	K ₂ O	Na ₂ O	CaO	MgO	Fe ₂ O ₃	P ₂ O ₅	SO ₂	Cl.
8th day, in mg.	504	224	188	27	1.1	160	48	358
70th day, in mg.	207	141	305	42	1.0	280	50	274

Surprise may be felt that the introduction of Na₂O and Cl on the seventieth day is much smaller than on the eighth day, while there is hardly any difference in the values for K₂O, Fe₂O₃, and SO₂. The reason is that 100 Gm. milk on the eighth day of lactation contains at least 0.3

Gm. ash, while milk on the seventieth day contains hardly 0.2 Gm. ash. The amount of Na_2O and Cl diminishes very much, and the amount of K_2O , Fe_2O_3 , and SO_3 decreases rather considerably, but this loss is equalized by the increased amount of milk. The milk of both periods contains the same amount of CaO , MgO , P_2O_5 , or of those ash constituents which are concerned in the formation of bone. Here the increased amount of milk is of benefit. The reason why the fixed alkalies and Cl are diminished is not known.

Table 7 shows that about 50 per cent, of the ash introduced into the body is retained. In regard to the individual constituents, it can be stated with certainty that less than 50 per cent. of the introduced fixed alkalies and Cl are retained, and more than 50 per cent. of the introduced CaO , MgO , P_2O_5 , and Fe_2O_3 . At present, it would be unsafe to give more detailed data, since the amounts entering into consideration are rather small and the difficulties of the analyses are therefore great. It may be mentioned that in speaking about organic substances, "mineral" and "ash" constituents are by no means identical.

The metabolism of the *artificially fed infant* does not differ in principle from that of the breast-fed, and it approaches the latter the nearer to the natural and the more rational the artificial feeding. The bottle-fed infant secretes more urine on account of the increased introduction of liquid, and the amount of feces is larger, since more food is given and the food is not utilized so well. The requirement of calories is increased, as the baby under certain conditions is more restless and has to perform more work during digestion. In individual cases, the metabolism of the artificially fed infant differs so much with the different methods employed, that an exhaustive presentation of the subject here would lead us too far. For instance, feeding large quantities of slightly diluted cow's milk with a small addition of lactose causes a strong excess of casein and ash, particularly of CaO and P_2O_5 , and the N and ash content of urine and feces are consequently increased. When more diluted cow's milk, with perhaps an addition of fat (cream) and lactose, is fed, the urine and feces contain quantitatively but not qualitatively more nearly the same amount of ash and nitrogenous substances as in the case of a breast-fed infant, and so on.

The healthy artificially fed infant receives a larger amount of food than does the breast-fed infant, at least unless the desire of the infant for a larger amount of food is denied with great consistency. If the infant remains perfectly healthy in spite of an excessive amount of food, to disregard entirely its desire cannot be advised. When the food requirement is much exceeded without injury to the infant, there will be a gain in weight. But the increased peristalsis, the more abundant excretion of urine and feces, etc., render the infant always decidedly more restless, and it cries more than the breast-fed infant of the same

age. During the first weeks of life, the breast-fed infant generally sleeps when not occupied with nursing; later on it lies quietly cooing, etc. Formerly, when infants were much overfed, physicians could not understand how to account for the disposal of the food introduced. For neither the gain nor the solid substances of urine and feces could come anywhere near the amount introduced. The substances are oxidized and leave the body by means of the gaseous metabolism, and their combustion furnishes the energy required by the increased peristalsis, the restlessness, and the crying. This agrees with the demands of the theory and has been directly demonstrated in the fourth individual experiment of Heubner and Rubner.

The metabolism of the breast-fed infant in the first week of life has to be considered briefly. This has been and still is a subject which brought forth many erroneous explanations and conceptions. During the very first days of life the infants suffer from the pressure on the head and brain experienced during birth; they sleep a great deal and do not require much food, wherefore they do not drink much even from the breast of a wet-nurse with abundant milk supply. An infant can only with great effort obtain a small amount of colostrum from the mother's breast. The metabolism is connected with a considerable loss of body weight (about 200 Gm.) (7 ounces) up to the end of the third day, after which the infants are several days in a state of convalescence.

The occurrences in these first three days may be illustrated by the following observation of Camerer (the elder) which is the most complete that has been made up to the present time. It was made on a girl with a birth weight of 3370 Gm., who was the fifth child, and who was nursed by her mother.

TABLE 14.

	Amount of milk.	Losses.		Insensible perspiration.	Sum of losses.	Changes in body weight.
		Urine.	Feces.			
1st day.....	10 Gm.	48 Gm.	51 Gm. meconium.	96 Gm.	197 Gm.	— 187 Gm.
2nd day.....	91 Gm.	53 Gm.	26 Gm. meconium.	79 Gm.	158 Gm.	— 67 Gm.
3rd day.....	247 Gm.	172 Gm.	8 Gm. feces.	85 Gm.	260 Gm.	— 13 Gm.
4th day.....	337 Gm.	226 Gm.	8 Gm. feces.	92 Gm.	321 Gm.	+ 16 Gm.

During the first twelve hours of the second day there was no secretion of urine at all. The urine of the first and second days is concentrated and particularly rich in uric acid (see p. 387); from the third day on, it becomes more dilute and similar to the urine of later infancy. The insensible perspiration may be estimated to consist of about 15 per cent. carbon and 85 per cent. water vapor. On the first, second, and third days we would have an excretion of carbon amounting to 15 Gm., 12 Gm., and 13 Gm., respectively, while 0.6 Gm., 6 Gm., and 18 Gm. carbon were introduced. In the first days about 20 Gm. of carbon (C

of urine and feces included) are lost, which would mean the decomposition of about 25 Gm. body fat. The loss on Na containing body substance is much less, since the colostrum is very rich in proteids. It may be calculated that nearly 1 Gm. proteid is introduced on the first day and 6 to 7 Gm. on the second and third day. On the second day, therefore, a gain of body substance is to be expected, while there may be some loss on the first day. The loss of body weight on this first day of about 190 Gm. is composed of 99 Gm. meconium and urine and about 100 Gm. insensible perspiration, against which there is an introduction of 10 Gm. milk. Some decomposed body substance has been passed with the urine, but this cannot be calculated, since at birth a certain amount of preformed urea, etc., is found in the tissue juices and in most cases already in the bladder. But these figures show that even on the first day of life—a veritable day of starvation—the loss on the said body substance cannot be more than a few grams of fat; the main constituents of the loss are meconium and body water (see Table 1, p. 362).

The infant sustains the weight on the third day with about 200 Gm. milk; on the fourth, fifth, and sixth the weight increases markedly on an average of 350 Gm. milk. This behavior, and the erroneous opinion that every increase of the infant's weight means growth, led to the conclusion that a sufficient growth could be obtained later on with some such "minimal quantity of food"; this would correspond to an introduction of 30 to 50 calories per kilo body weight.

Such ideas, which are justified to a certain degree under pathological conditions, are absolutely out of place in regard to the healthy infant. We have seen that an infant may increase in body weight for a short time, even if moderately underfed, but this entails a loss of body fat, and a normal fat content (about 12 per cent. of the body weight) is necessary to sustain health. The gain of the infant from the fourth to the eleventh day is mostly composed of water and a little fat, as in all cases of convalescence. On the eleventh day it may have reached the birth weight on water and fat, and beside it may have used up about 4 Gm. N, which is equal to 30 Gm. body substance. From now on, the gain and fat will be smaller.

GENERAL CONSIDERATIONS CONCERNING THE NOURISHMENT OF THE INFANT

The foetus receives all substances necessary for its development through the mother's blood, and these substances enter the blood of the foetus without coming into contact with the digestive tract. The organism of the infant is separated from that of the mother at birth, and with this act a sudden and great change takes place. The little newcomer has to dispense with its protecting cover and is exposed to the dangers and injuries of its new surroundings, particularly to a consid-

erable undercooling. The needed oxygen has to be supplied through the hitherto inactive lungs and blood circulation. The food requirement increases very much, and the food introduced from without has to be prepared for utilization through the activity of the stomach and the intestine. The great majority of newborn infants are exhausted by hardships undergone at birth, and some are injured considerably. It is really not surprising, therefore, that of those who die within the first month of life about one-fourth do not survive the first day.

A great many newborn infants would not have many chances to escape, emerge victorious and unscathed from this serious struggle for life, if nature had not provided a food perfectly adapted to their requirements and their powers of digestion. For in spite of the separation of the bodies of the infant and the mother, the mother is capable of providing the nourishment for the newborn for several months, in the form of mother's milk. It is well known that the great majority of infants receiving the natural food from the mother's breast do well and relatively rarely suffer from disorders of nutrition. On the other hand, the mortality among artificially fed infants, particularly during the first month of life, is exceedingly great, and this mortality is due in great measure to disorders of nutrition. The health of many of the surviving infants may be unmistakably impaired for a longer or shorter period of time, so that it is easy to distinguish the breast-fed from the bottle-fed simply by their appearance. This empirical observation finds its explanation in the investigations of more recent years. It was shown that the milks of various animals differ considerably. Bunge expresses the view that for suckling purposes the milk of one animal can never completely replace that of another. Although some of Bunge's assumptions have been shown to be untenable, his views as a whole are justified and have received confirmation through the biochemical investigations of recent years.

While there is no doubt that *the mother's milk is the best food for the newborn infant*, there are several reasons why, in Germany at least, only a minority of infants receive breast-milk exclusively. The majority of infants are raised partly or entirely artificially. The noblest and most satisfying duty of the physician and hygienist is to alter this state of affairs, and to insist that as many newborn infants as possible shall receive the benefit of the breast-milk. Within the last few years a certain improvement has crowned the efforts undertaken in this direction, and to show how much can be done, the following example is cited. Herdegen, in 1882, inquired into the nursing ability of the women delivered in the school of midwifery in Stuttgart. These women remain in the institution for twelve days after parturition. Only 23 per cent. were able to nurse their infants. In the same institution in 1904 Martin made observations regarding the same problem, and the interesting fact was found that nearly 100 per cent. of the women were able to nurse their

infants. The material was furnished in both cases from about the same classes. No doubt the ability to nurse did not increase to such an extent in this short period, and it must be assumed that the figures of Herdegen did not give a true picture of the conditions. The number of women able to nurse increased only because it is insisted on more vigorously now that all women able to nurse must actually do so. This example illustrates clearly, moreover, how little we can rely on statistics, particularly on old ones, with regard to the ability to nurse, and how careful we have to be in their use. A comparison of the figures given by Herdegen with those of previous years form the mainstay for the arguments of Bunge concerning the increasing inability of women to nurse.

Many physicians do not show the necessary interest and understanding in the effort made to increase maternal nursing. It frequently happens that obstetricians pay more attention to the welfare of the mother than of the child, and do not lay sufficient stress on nursing; or they even advise against it, without having any valid reason to offer. Not infrequently, physicians claim that artificial infant feeding has reached such a high degree of perfection that it is not inferior to the breast-feeding, while in certain respects it is even more convenient to carry out. It must be conceded that at present artificial infant feeding can be carried out on understood principles and with the prospect of good success. In former times it was a dangerous experiment, in the execution of which the physician had to grope in the dark or had to rely on trials for which there were no due indications. But often artificial feeding does not succeed in spite of all efforts, to say nothing of the numberless cases where the want of care or insufficient understanding is followed by severe impairment of health or even death.

On the other hand, the fanatic advocates of nursing go entirely too far when they affirm that artificial feeding cannot be but injurious, and that the species is bound to degenerate even if some children seem to grow up strong and healthy, so that after a number of generations the evil consequences will be distinctly manifest. Nevertheless, artificial feeding can never completely replace the breast-feeding, for it cannot be carried out with the same certainty of success and it requires a greater expenditure of time, labor, and money.

NUTRITION AND METABOLISM OF THE BREAST-FED INFANT DURING THE FIRST WEEK OF LIFE

The newborn infant, exhausted by the hardships undergone at its birth, the cleansing, bathing, etc., requires rest more than anything else, and it soon goes to sleep, waking up only rarely and for short times during the first twelve to twenty-four hours. During this time no food is required. Should it begin to cry or become very restless after the bath and rearrangement of its couch and clothing, it may be placed at the

breast ten to twelve hours after birth. Whether the infant obtains any food at all, depends on its aptitude for suckling and on the contents of the breast. As a rule only very small quantities are obtained even with strong suckling and when the breasts contain a sufficient supply. The amounts of milk taken are determined by weighing the baby before and after feeding. On an average 10 to 20 Gm. milk are taken in one or two meals during the first twenty-four hours. In the second twenty-four hours about 90 Gm. milk are taken in four to six meals. From this time on the following average values were obtained:

TABLE 15.

	3d day.	4th day.	5th day.	6th day.	7th day.
Amount of milk in 24 hours.....	190	310	350	390	470
Number of meals.....	5-8	5-8	5-8	5-8	5-8

Deviations from these average values in either direction are observed, corresponding to the differences in the milk supply, strength of the baby, etc. But while little harm is ever produced by taking an insufficient amount of milk during this period, overfeeding may lead to digestive disturbances which may last through a long period of time, and are sometimes corrected only with difficulty.

It is undesirable to give the breast more than eight times during twenty-four hours. Every two hours is a custom still frequently in vogue, even if the secretion of milk is insufficient and the baby is weak. It would be necessary on a two hours' interval to interrupt the sleep too frequently. The best method is to put the baby to the breast six or seven times in twenty-four hours.

In the first days post partum, particularly with primiparæ, no milk (or rather colostrum) or very little, is available. Anxious mothers or nurses are very easily inclined to give additional feeding under these circumstances, even when the infant is perfectly quiet. When the baby becomes gradually more and more restless, and when hardly any milk is produced on the second or even the third day, in spite of frequently repeated efforts at suckling, many physicians doubt the possibility of feeding at the breast and deem the transition to artificial feeding necessary. Later investigations have proved that in many cases the lactation is not established before the second half of the first week, and may in some instances occur even later. The lactation in such cases takes a perfectly normal course. The absolutely necessary condition is that it shall be excited by a rational stimulus, which is to be found only in putting the baby frequently to the breast, and in taking care that the breast is emptied as completely as possible. To leave off too soon trying to nurse means nothing less than to deprive the infant of the irreplaceable mother's milk. The proposal of Czerny and Keller during the

first three or four days not to offer any other nourishment to the infant but the breast, can hardly be accomplished in practice, particularly when the infant is restless, as the authors themselves recognize. They justly state that as a rule the newborn infant will not suffer any serious injury, even if it does not receive any food during the first two or three days, yet it does not appear necessary or desirable not to give anything. While it may not be absolutely necessary to give any food, it will be rational to provide a certain quantity of water, because, if none is given, an excessive concentration of the urine may cause a lesion of the kidneys, and the contents of the large intestine may become inspissated, with evil result. It seems advisable, therefore, to give small quantities of water from time to time from the second day on, or, if the infants are very restless, even on the first day. Boiled water or thin tea sweetened with a trace of saccharin is preferable. Keller justly advises against the use of sugar water. On the second and third days 15 to 30 c.c. ($\frac{1}{2}$ to 1 ounce) saccharin water are given four to six times, and later correspondingly more, in which way the administration of artificial food can be delayed without any uneasiness until the fifth or sixth day. Should the secretion of milk be delayed beyond this time in spite of good suckling, as a rule artificial feeding must be started. The writer observed a case, where in spite of an entirely insufficient milk supply, a strong infant did not receive any additional feeding with exception of water until the 14th day, when the lactation became established and continued for four months.

The quantity taken at a single meal is subject to very wide variations. It is dependent on the milk supply, the state of the nipple, the strength and food requirement of the infant, and the number and duration of the meals. As a rule, the largest meal is taken in the morning, after the long night interval.

Healthy infants nurse until they stop of their own accord. An infant properly placed at the breast takes its meal in five to ten minutes as early as the second half of the first week, if the milk is good. The majority of infants, however, take fifteen to twenty minutes for even a small meal. It is of no use to leave an infant at the breast much longer, because the quantity of milk taken in a unit of time becomes less and less the longer the duration of nursing. For instance, a somewhat older baby observed by Feer drank during the first 5 minutes 112 Gm. milk, during the second 5 minutes 64 Gm., and during the third 5 minutes 10 Gm. Very weak infants, who are easily fatigued by the work of suckling and have to rest during the meal, may be left at the breast for 30 minutes. But the scales ought to be employed in order to find out whether the lengthening of the meals actually increases correspondingly the quantity of food received. Such infants, and infants too lazy to drink, may occasionally be induced to continue nursing by slight

motions. The amount of milk taken may be roughly estimated by observing the frequency of the act of swallowing.

Although it is not advisable to arouse an infant from its sleep in order to feed it, yet it is very desirable to adhere to regular hours for feeding, and it is remarkable how much can be accomplished by education. The infant should be trained to take its meals in the first week about every three hours in day-time, while at night an interval of from five to eight hours should be interposed. If the infant is very weak and drinks little at a time, the intervals in day-time may be shortened to two and a half hours, but they should never be decreased any further.

Primiparae have certain difficulties in putting the baby to the breast for the first time, even when the nipples are well developed and the baby is not awkward. These difficulties are mostly caused by an improper position of the child (retroflexion of the head, occlusion of the nose), or by an insufficient introduction of the nipple into the mouth. But the young mother soon acquires the necessary experience and skill. Should the nipple be poorly developed, it is more difficult to make corrections. In light cases it is only necessary to draw out the nipple as much as possible with a breast-pump before the baby is put to the breast; or the baby receives, beside the nipple, a part of the areola; or a nipple shield may be employed. If the procedures are carried out skilfully we are likely to be successful more frequently than we are inclined to believe. In cases of inverted nipples, the possibilities of nursing at the breast are very small and the nipple shield will only rarely be of any help. The milk may be drawn with the breast-pump and the baby will receive the benefits of the mother's milk in this manner.

The normal baby nearly always becomes very skilful the second or third time it is put to the breast and very soon may show a certain individuality in nursing. One drinks more rapidly, the other more slowly; one prefers a more reclining, the other a more sitting position. Pfaundler justly emphasizes the fact that a careful observation of such peculiarities may prove of considerable importance in the growth of the baby. The introduction of the nipple into the mouth of the eagerly "searching" baby immediately calls forth motions of suckling. The lip and the tongue enclose the nipple hermetically, and while the mouth cavity is closed behind, the sinking of the lower jaw, the floor of the mouth, and the tongue creates a diminished pressure in the cavity of the mouth. Pfaundler has shown that this does not cause the milk to enter the mouth. Only the filling of the sinus lactei is accomplished and the nipple is held firmly. The suction itself does not remove the milk from the breast at all, and the infant receives the milk through compression of the nipple in closing the jaws. If the secretion of the milk is abundant, the suckling is immediately followed by a motion of swallowing. But if

only a little milk enters the mouth by one act, the swallowing occurs only after repeated suckling.

The work performed in suckling is not inconsiderable; the pressure of suction amounts in the first week to about 10 to 20 cm. water, and in older infants to 70 cm. or more. The infant thus performs a rather considerable amount of work in taking its food, which work fatigues it at the proper time and protects it against excessive feeding. At the same time it stimulates the secretion of the gastric juice (Pfaundler), either by an increased secretion of saliva, or, more probably, through special reflexes.

During the first days of life the infant should suckle both breasts at each meal, but toward the end of the first week strong babies should receive only one breast. The breast is then emptied completely and the infant receives the benefit of the last milk, which is richer in fat. Emptying the breast is very favorable also for the production of milk. In cases of weak infants and insufficient milk supply, the production of milk can frequently be increased to meet the demand by putting a strong infant to the breast. If an infant receives only one breast at a meal it has to perform more work in suckling, and strong infants can do this without injury. But weak, easily fatigued, and premature infants should always receive both breasts if the milk production is not very abundant.

Before nursing, the mother should express some of the milk, because the first milk is not sterile, on account of the entrance of bacteria into the milk-ducts. It is important to cleanse the nipple with boiled water before and after nursing. It is neither necessary nor desirable to use antiseptic fluids. The mouth of the infant should not be washed before or after nursing, since, according to the investigation of Epstein, this may easily lead to injuries of the mucous membrane, with their sequelæ. But the exterior parts of the mouth, and particularly the corners, must be kept clean.

The secretion of the mammary gland in the first days post partum, the colostrum, differs very much in its composition from the milk of other periods. Its physiological significance is pointed out in the chapter on the milk, and here only the essential difference from the milk proper, which lies in the high content of albumin, will be emphasized. According to Camerer (the elder), the milk during the first week is as follows:

TABLE 16.

	1st day of life.	2d day.	3d day.	4th day.	5th day.	6th day.	7th day.
N	0.28	0.66	0.99	1.14	1.12	1.25	1.14
Proteid	1.6	3.8	5.7	6.3	6.1	6.8	6.6
Fat	1.2	5.1	7.8	9.9	11.2	12.5	14.5
Sugar	1.2	7.1	12.9	16.8	19.4	21.7	28.6
Ash	0.1	0.5	0.7	0.9	1.0	1.1	1.3
Water	25.3	111.6	200.1	263.1	299.2	334.5	385.5
Unknown substances	0.6	1.9	2.8	3.0	3.1	3.4	3.5

Against this introduction of food there is the excretion in the urine, feces, and insensible perspiration. (In regard to the latter, see Table 8, p. 373.)

THE URINE IN THE FIRST DAYS OF LIFE

The secretion of urine during the first day of life amounts to only a few cubic centimetres; sometimes none is voided. On the second and third days, still very little urine is voided, but here the amount is distinctly dependent on the ingestion of fluid. From now on the amount secreted depends mostly on the amount of fluid taken, so that 60 to 70 c.c. urine are secreted for each 100 c.c. milk taken. Accordingly, the amount of urine secreted in 24 hours may be calculated as 200 c.c. on the fourth day, and as 300 c.c. on the seventh day. The frequency of urination varies greatly, and may amount to 20 or 25 times in 24 hours. Corresponding to the small quantity, the concentration and the nitrogen content of the urine are relatively great in the beginning. The specific gravity varies between 1.008 and 1.012; it diminishes rapidly to 1.003 to 1.004, to remain for months at this level. The color of the urine is intensely yellow in the first days, and the reaction is acid; even the freshly voided urine is somewhat turbid, owing to the precipitation of urates and epithelial cells from the urinary passages. In the second half of the first week the urine becomes clear and its color a light yellow, corresponding to the low concentration.

The chemical examination shows in numerous newborn infants (in all, according to Flensburg) the presence of albumin (nucleoproteid). Toward the end of the first week, in the majority of cases, this surprising phenomenon ceases, but sometimes a trace of albumin may be found for weeks. The morphological elements to be found are epithelial cells from the urinary passages, leucocytes, amorphous or crystalline uric acid, hyaline, granular, and epithelial casts, brown masses of ammonium urate in the shape of casts, and occasionally epithelial cells from the kidneys. While the number of casts becomes very small on the third day and these soon disappear completely, leucocytes, epithelial cells from the urinary passages, and large quantities of urates are still found to the end of the first week.

A complete agreement of opinion in regard to the causes and the meaning of this albuminuria of the newborn has not been reached, but it seems probable that it is not due to any morbid process in the mother or the child, but is a physiological phenomenon. The changes in the circulation, in the quality of the blood, and in the metabolism from embryonic to extra-uterine life may be regarded as the foremost causes.

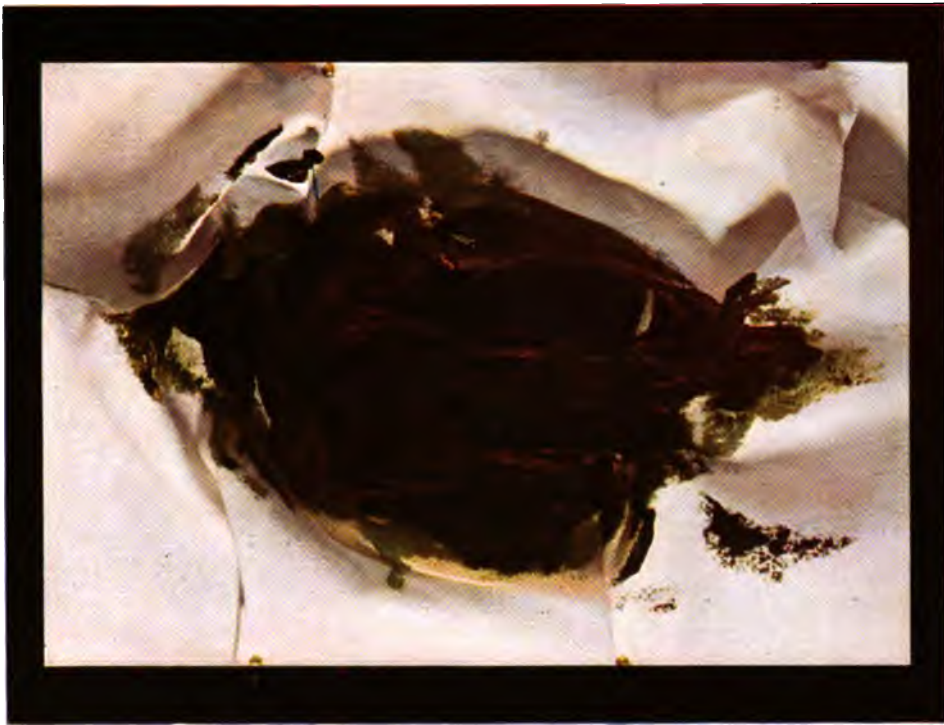
Closely connected with this are the uric acid infarcts in the kidneys of the newborn, first described by Virchow. In the great majority of infants dying on the second or third day, a large number of yellowish

red striæ are to be seen microscopically in the pyramids, starting from the middle or occasionally from the border of the medullary substance, and converging like the straight urinary tubules toward the papilla. Microscopically, the straight uriniferous tubules are filled with cylindroid pieces of yellowish brown and partly gray color or with granular masses. They are composed of ammonium urate or crystals of uric acid, imbedded in a hyaline-like, partly unformed, partly cylindroid substance. Flensburg, after careful investigations, explains the origin of the uric acid infarct as follows (cited from Czerny and Keller, *Des Kindes Ernährung*, etc., part I, p. 189): "The primary part of the infarct is formed by the proteid-like substance, which is secreted in the convoluted tubules of the kidney during embryonic life. When during the first days of life the kidneys secrete a very concentrated urine rich in urates, the hyalin-like elements of the infarct block its way and prevent its passage. The almost insoluble ammonium urates, present in large quantities, are deposited on this hyalin-like substance like salts crystallizing from a concentrated solution on a string." Flensburg and Reussing explain the increase in the excretion of uric acid during the first week by the supposed hyperleucocytosis of the first days of life. A large number of leucocytes perish, and furnish the material for the increased production of uric acid, according to the well known investigations of Horbaczewski. Although it is not certain that the concentration of the urine and its large content of uric acid in the first days of life constitute the only causes of the uric acid infarct, it may be permissible to assume that the infarct is not due to a morbid process, but represents more or less a physiological phenomenon. This assumption is strengthened by the fact that the kidneys frequently do not show any pathological changes, in spite of the presence of an infarct.

From the foregoing it will be readily understood that the formation of a uric acid infarct is rarely to be found in still-born infants or in infants dying a few hours after birth, and this fact has frequently been made use of in forensic medicine. Most frequently the infarct is observed in infants dying in the second half of the first day or on the second or third day; it is seen much less frequently on the fourth and fifth day, and from then on only very exceptionally. Taken as a whole, a uric acid infarct is found in more than half of the infants between two and fourteen days of age, on which a post-mortem is performed (Hecker).

The fact that the albuminuria occurs at the time when the uric acid infarct is observed, points to a connection between these two manifestations. Virchow, and particularly Hofmeier, called attention to this connection. It is probable that as a rule the mechanical or chemical irritation caused by the infarct gives rise to the albuminuria, and the appearance of casts, etc., might easily be dependent on the same causes. But sometimes the course of these two processes shows certain differences.

PLATE 3.



a



b

a. Meconium. b. Transition from meconium in a breast-milk stool on the third day of life.

For instance, the infarct is seen in only a small number of cases during the second week of life, while albumin is still excreted though in diminished quantities. Therefore, as Flensburg justly remarks, the presence of the infarct is not sufficient to explain the occurrence of the albuminuria. Further investigations are needed to decide this question finally.

MECONIUM AND FECES OF THE BREAST-FED INFANT

The first evacuation of the bowels occurs only a few hours after birth, or, if the labor is protracted, it may occur during birth. This is a dark brown, mostly odorless, homogeneous tarry mass called meconium (see Plate 3). Its sources are swallowed amniotic fluid, secretions from the intestinal tract of the embryo, and intestinal epithelial cells. Microscopically, lanugo, cholesterin, fat globules, platelets of epidermis, epithelial cells, and peculiar greenish yellow, oval or rounded bodies, so-called meconium corpuscles, are found. The opinions concerning the origin of the latter are not quite uniform, but it is probable that they are derived from epithelial cells of various origin, which while passing through the intestines shrink, break up into small pieces, and become strongly stained with bile.

The total amount of meconium is about 60 to 90 Gm., (2-3 ounces) and infants taking the breast well dispose of most of it in small separate portions on the first day and in the first half of the second day. But, particularly in cases of pronounced underfeeding, meconium may occur in the feces as late as the fourth day. Camerer observed with the first excretion of meconium a small grayish white glassy mass. This is composed mostly of mucus, detritus, and epithelioid cells. Its presence may be of some significance forensically, since it proves that no meconium had been voided previously.

According to the investigations of Zweifel, F. Müller, and others the meconium contains 70 to 80 per cent. water. The dry residue contains about 5 per cent. nitrogen, 15 per cent. ether extract, and 4 to 5 per cent. ash. Hydrobilirubin is absent; the presence of bilirubin, biliverdin, glycocholic acid, taurocholic acid, and cholesterin can be demonstrated.

In most cases the character of the feces changes in the course of the second day (see Plate 3). The discharge from the intestines acquires a lighter color and becomes softer, and after a short time the pure milk stool makes its appearance. Its color is light yellow to golden yellow, the consistency is pasty, the odor is agreeable and the reaction is acid. The composition is not quite uniform, but nearly always numerous small white pieces, gas bubbles, and mucus, particularly during the first week, are imbedded in the yellow mass. During the first week the daily passages number two to four, later one to two. When a normal amount of food is introduced about 2 to 3 Gm. feces are excreted for each 100 Gm.

mother's milk, and about 4 Gm. solids for 100 Gm. milk solids. In cases of overfeeding the resorption is not so good. During the first week fresh milk stools contain twenty to twenty-five per cent. dry residue, later fifteen to twenty per cent.; of this ten per cent. are inorganic substances,—one-third of which are calcium salts,—and ninety per cent. organic substances (mainly bacteria). Beside the ash, the dry residue contains about twenty to thirty per cent. fat, soap, and free fatty acids, and four to five per cent. nitrogen.

The microscopical examination of the fresh milk stool reveals the presence of numerous bacteria, fat globules of varying sizes, crystals of fatty acids, salts of fatty acids (soaps),—partly in crystalline form partly amorphous,—crystals of cholesterin and bilirubin, more or less changed epithelial cells, and masses without structure. The small whitish clumps, which can be seen macroscopically, have long been regarded as casein. But most of these consist of calcium soaps and fat globules, and some of them are made up of bacteria, probably knitted together by a proteid-containing substance.

The quality of the feces does not change materially under normal conditions during the whole period of lactation (see Plate 38). Frequently in the first months, apparently perfectly healthy infants have passages which are not uniform in consistency, and which present the appearance of chipped beef and are slimy or watery. As a rule, these manifestations disappear quickly without any therapeutic measures. But these cases very probably represent light pathological conditions, which may be due to changes in the quality of the food (according to Gregor variations in the fat content of the milk), or may be caused by light digestive disorders otherwise not demonstrable.

NUTRITION OF THE BREAST-FED INFANT FROM THE SECOND WEEK TO WEANING

In the beginning of the second week, as a rule, the disturbances in the infant incident to its birth have disappeared. The intestinal tract has adapted itself to the new conditions and lactation is well established. Under normal conditions the technic of feeding changes very little during the next months, and this is simple enough, since we may grant the healthy infant its freedom in regard to the amount of its meals, which it usually takes with a surprising regularity. A certain control is necessary, nevertheless, particularly the continued observation of the weight and of the number and character of the passages. This enables us to recognize without difficulty excessive or insufficient ingestion of milk and peculiarities of its chemical composition; and that mostly at a time when severe injuries are still to be avoided.

Observations on numerous healthy breast-fed infants of the same age have shown that the amount of milk taken in 24 hours may vary to

a certain extent with the individual infants, and within short intervals of time, corresponding to the difference of the body weight, the activity the composition of the milk, etc. Variations and differences of several hundred grams may occur, but as a rule the values are not far removed from an average figure. The average amount of milk taken in 24 hours is:

TABLE 17.

	Middle of						
	2d week.	4th week.	8th week.	10th week.	14th week.	17th week.	20th week.
Grams	500	600	800	820	850	870	900

The following rule furnishes approximate figures, sufficiently accurate for all practical purposes: To insure proper growth and development the infant requires daily an amount of mother's milk equal in the first quarter year to one-sixth, in the second quarter year equal to one-seventh, and in the third quarter year equal to one-eighth to one-ninth of its body weight.

While the absolute amount of milk taken increases, at first rapidly and later slowly, the relative amount decreases in the second quarter and still more in the third. The reasons for this phenomenon have been given above.

The absolute and relative ingestion of proteid may be calculated from the figures given in Table 18, assuming that the milk contains 1 per cent. proteid.

TABLE 18.

	2d week.	4th week.	8th week.	10th week.	14th week.	20th week.
Absolute grams proteid taken in 24 hours.....	5	6	8	8.2	8.7	9
Grams proteid per kilo body weight...	1.5	1.5	1.7	1.6	1.5	1.3

A similar calculation of the quantities of fat and of milk-sugar is of less interest and offers more difficulties. The knowledge of the amount of energy supplied to the infant is more important. Exact knowledge in an individual case requires a careful calculation of the amount of milk taken in 24 hours, and a direct determination of the combustion value of the milk or an analysis of the milk. It is sufficient for practical purposes to calculate the energy supplied, taking as a basis a content of 700 calories per litre. The energy supplied per kilo body weight (energy quotient, according to Heubner) amounts to about 110 calories in the first quarter, 100 calories in the second and 90 calories in the third.

Should the energy quotient drop much below these figures, we can not expect a proper development of the infant, except under particular circumstances. Should an infant not thrive in spite of an apparently

normal supply of energy, it is probable that the composition of the milk taken deviates from the average composition of milk, and particularly that the fat content is low. Should an infant thrive in spite of an apparently insufficient supply of energy, the milk very probably is extraordinarily rich in fat.

During the first three months six to seven meals are taken in 24 hours, and during the second three months five to six, so that the intervals between meals are first three hours, and later three and a half to four hours in day-time, with corresponding intervals at night. Great difficulties are frequently encountered in the institution of this arrangement, because of the desire and the custom of many mothers to nurse the infant more frequently. Infants accustomed to more frequent meals will at first express their dissatisfaction distinctly. But a rigorous adherence to the less frequent meals and the long intervals is of the utmost importance, in view of the harm that can easily be done by too frequent meals with excessive ingestion of food.

The average size of a single meal, which varies considerably in the course of a day, is:

TABLE 19.

2d week.	4th week.	8th week.	10th week.	20th week.
75 Gm.	100 Gm.	130 Gm.	150 Gm.	170 Gm.

WEANING

The appearance of the first incisors, generally at about the seventh month, indicates that the time has arrived when the infant may pass gradually from the exclusively fluid diet to food somewhat more solid. Occasionally the teeth furnish the immediate cause for weaning, if the infant bites the nipples while suckling and makes them sore. If the production of milk remains sufficient some mothers continue to nurse their babies up to one year or even longer. Observation has demonstrated that such infants do not thrive particularly well, but look pale and puffed up. This may be perhaps due to the poverty of the mother's milk in iron, and such a prolonged nursing is not of benefit to the mother. An infant should therefore not be exclusively breast-fed longer than nine or at the most ten months. Very frequently, however, the nursing has to be interrupted prematurely for some reason, as premature cessation of the secretion, disease of mother or child, advanced pregnancy, or external circumstances. The earlier this happens the more dangerous it may be for the baby. If possible, the transition should be accomplished only gradually and at first the bottle should be given alternately with the breast. In giving artificial nourishment, which must be regulated according to the age, weight, and state of health of the infant, it must be

remembered, that the infants obtain their food without effort and do not become fatigued, and that they are thus readily exposed to the dangers of overfeeding. Care must therefore be exercised in the beginning, that the food is not too abundant in quantity and quality. As soon as the infant has adapted itself to the new food a possible temporary standstill of weight can easily be made good.

If possible, the infant should not be weaned during the hot season or when the dentition is very active. The weaning must be done very carefully and slowly. It is advantageous in the course of three weeks to replace one breast-feeding after the other by the bottle, giving the bottles between breast-feedings as far as possible. It is then possible to return to the breast if the artificial food does not agree with the baby. In most cases the weaning succeeds in this way without much difficulty. But babies always respond by severe intestinal disturbances to the ingestion of artificial food. Here much patience and many trials are required to reach the goal. In cases which have to be weaned at the proper time, diluted cow's milk is generally given at first (best beginning with two parts milk and one part water), increasing to greater concentration after a few days, until soon whole milk is given. According to the infant's taste and the state of digestion, something may be added to the milk; in most cases cane sugar or milk-sugar, or "Nährzucker" (a mixture of maltose and dextrin, introduced by Soxhlet) will suffice. In difficult cases, suitable higher dilutions are given, in very small amounts, and the increase is very gradual. Some infants refuse to drink from the bottle, and the author observed a case where the milk could not be given except with a spoon.

Infants weaned at the right time should soon receive some additional food beside cow's milk. Thin beef broths with rice, barley, or tapioca are particularly to be recommended, and gradually yolk of egg is added. Later the fare may be enriched by cocoa, zwieback, gruels, prepared from the known flours for children, with milk or bouillon, and toward the end of the first year tender vegetables, finely chopped meat, and stewed fruit are given. The amount of milk per day should not much exceed 1 litre (1 quart) at this period.

MIXED FEEDING (ALLAITEMENT MIXTE)

When the secretion of milk is not sufficient for the demands of the infant from the time of birth or later, or when other reasons (for instance, external circumstances) do not permit the baby to have the required amount of mother's milk, it becomes necessary either to give artificial food in addition or to raise the infant on the bottle entirely. In Germany until a short time ago the latter method was generally adopted, because it offers certain advantages, due to its greater uniformity, and to the fact that many children do not like to nurse after becoming

acquainted with the more convenient drinking from the bottle, which fact exercises an unfavorable influence on the secretion of milk. Fortunately, circumstances have altered since French observers have shown that mixed feeding may be carried out in many cases with good success. It is surprising how frequently the secretion of milk can be kept up to a satisfactory amount for many months, even when the mothers are not able to nurse the baby during the day-time. It is important to make the drinking from the bottle more difficult by providing a nipple with fine holes (the use of suction tubes, or of a suction apparatus which can be regulated, is not advisable), and to make the baby more eager for the breast by relatively long intervals between bottle- and breast-feedings.

The quantity and the composition of the additional food is regulated according to the age, weight, and state of health of the infant (see the chapter on Artificial Feeding), and furthermore depends on the amount of available mother's milk. This has to be determined carefully. Under certain conditions one bottle-feeding will suffice, while in other cases perhaps only one breast-feeding can be given in 24 hours. The different meals should alternate as far as possible and should be separated by corresponding intervals; if the secretion of milk is very scanty and the infant is strong, the breast and the bottle may be given at the same meal, but under such conditions the amount of mother's milk taken should be determined.

During the first weeks of life the mixed feeding is of the greatest importance. The younger the infant, the more valuable is each drop of human milk. In this period the majority of women are capable of nursing. Not infrequently it is even possible to return completely to the breast-feeding. Czerny and Keller emphasize this advantage, that by this plan the mothers may become convinced of their ability to nurse.

ARTIFICIAL FEEDING OF INFANTS

Artificial feeding, in distinction from natural and mixed feeding, means that an infant does not receive any mother's milk during a part or all of the period in which it should be nursed. The artificial feeding may be instituted immediately after birth, or after an infant has received the breast exclusively or partly for a longer or shorter time. While it is relatively easy to rear artificially a healthy infant five to six months of age, after it has been gradually weaned, experience has taught us that exclusive artificial feeding from the time of birth can rarely be accomplished without more or less severe disturbances, even when the greatest care is exercised. Fortunately this feat has only rarely to be accomplished, since the majority of infants receive the breast during at least the first days and weeks.

Naturally, the feeding at the breast serves as a prototype for the artificial feeding. The increasing knowledge of breast-feeding has made it

possible—as has been pointed out previously—to replace rough experimenting by more rational methods. As a result, the feeding has become more successful. The investigations of recent years have clearly demonstrated that mother's milk cannot be replaced, and the opinion which was still heard a few years ago, that it might finally be possible to obtain just as good results with artificial feeding, has been shown to be erroneous. It must be stated, however, that generally the results of artificial feeding, at least in infants who received the breast during the first weeks or months, have been very good, provided the artificial feeding was conducted carefully by competent persons. Faulty methods are more often responsible for bad results than is the artificial feeding itself. Ebert showed that the artificial feeding was faulty in 95 per cent. of the cases which were admitted to the dispensary of Heubner. It would be entirely erroneous to assume that all the difficulties could be overcome by slavishly imitating the breast-feeding, and that every new discovery in the field of natural feeding must be applied immediately to artificial feeding. Such errors have been made, and led, for instance, to the production of Dr. Rose's artificial milk. The correctness of the method to be used can be measured only by determining whether the results approach as nearly as possible those obtained by the natural feeding. The growth of the infant forms one of the most important and most striking indications of its nutrition. Regular determinations of the body weight and of the length are of the utmost importance in the control of artificial feeding, particularly during the first weeks of life and in delicate infants. Certainly the weighing must be done in a manner free from objections, and it is best to weigh at definite times, in the morning before feeding, without clothes or with always the same light clothing. It must be remembered that gain in weight is not always a sign of thriving.

A uniform method, which must be adopted in every case of artificial feeding, does not exist. This fact is frequently cited at present to prove our lack of sufficient knowledge, but it simply shows that an infant may be reared by different methods.

In presenting a metabolism balance of the breast-fed infant, the conditions necessary for a successful feeding were explained. Artificial feeding must so far approach breast-feeding that the digestive organs can dispose of the food without too great an excess of work, or at least that they are not injured by the food. It is necessary also that a sufficient amount be absorbed of those substances that are needed for the sustenance and growth of the body, and, moreover, the requirement of energy has to be met. Only the breast-milk fulfils all these conditions in an ideal manner, but different methods of artificial feeding may respond to the demands more or less. The choice of method must be based on a careful consideration of the individual case.

All methods have in common the use of milk derived from animals. This is a glandular secretion analogous to human milk which it approaches in its behavior. Cow's milk is most frequently used, and goat's milk is also used very much. With us, the ass's and the mare's milk, which are chemically more similar to human milk, do not play a rôle in artificial infant feeding.

Comparative investigations of human, cow's, and goat's milk have shown that while these kinds of milk have many properties in common, numerous differences exist, particularly of chemical-biological nature. For a more detailed account see page 305. Here only the difference of the proteid content will be pointed out.

Cow's milk contains about three times as much proteids as does human milk. The proteids of the latter are composed of about equal parts of casein and albumin, while cow's milk contains 7 parts of casein to about 1 part of albumin. Beside, there exist certain qualitative differences between the respective proteids of cow's milk and human milk, and these are perhaps responsible for the difference of certain qualitative reactions (precipitation of human casein in fine flocculi, of cow's casein in coarse curds; residue of pseudonuclein in digestion experiments with cow's casein, but not with human casein; different behavior with regard to the action of certain acids, etc.). The biochemical investigations have for the first time shown absolutely that human milk cannot be replaced by milk of animals. Although these results are of great significance, it is impossible at present to decide their practical importance, and it appears premature to use them for the explanation of the less satisfactory results of "unnatural feeding."

In contradistinction to human milk, which the infant receives directly and without manipulation, cow's milk is exposed to changes at the place where it is obtained and to further changes on its way to the consumer. These changes are of the greatest importance when the milk is to be used for the infant. Bacteria by their activity and growth affect the constituents of the milk. How these changes occur and how they can be avoided are discussed on page 317. The danger of bacterial contamination is very small, if the milk comes from healthy animals, properly kept and fed, and is obtained and kept with the greatest cleanliness and cooled as rapidly as possible, and delivered in this state. Under these conditions the milk may be used raw, provided it is further kept in such a way that the bacteria cannot grow at all or can grow only very slowly, and that the milk is consumed within one or at the utmost two days after milking.

All these conditions can be fulfilled much more readily when goat's milk is used. Goats are less subject to tuberculosis than are cows, and the danger of contamination of the milk is much diminished on account of the solid feces. Furthermore, the deteriorations caused by the deal-

ing with and the transportation of milk can be avoided with less difficulty, as many people can afford to keep a goat.

Such a milk, aseptically obtained and kept, seems to be the most suitable substitute for breast-milk, since it has not been exposed to the possibility of changes and has not lost its natural properties. Within recent years, many authors have expressed their preference for raw milk whenever possible, on account of its biological properties.

Heating is the most suitable method to inhibit the growth of bacteria. The total destruction of all the micro-organisms, *i.e.*, complete sterilization, cannot be accomplished with absolute certainty by simply boiling the milk. At first this was expected, but certain spore-bearing bacteria (particularly the bacterium described by Flüge), which occur very frequently in the milk, are not destroyed. Their continued growth causes dangerous changes in the milk, particularly by their action on the proteids. These bacteria are absolutely destroyed by a prolonged action of temperatures above the boiling-point. But by this the milk is changed to such a considerable degree that its use as food for infants is excluded, and a suitable milk, which will keep for a longer time, cannot be prepared in this manner.

Boiling the milk destroys all the micro-organisms, and particularly the pathogenic bacteria, with the exception of certain spores. Thus a partial sterilization takes place, which meets all the requirements of practice, provided the milk is consumed within twenty-four or at the latest forty-eight hours after milking. During this time it must be kept in such a manner that the germs which have not been destroyed cannot grow or can grow only very slowly, and that no new germs can enter the milk. The best way to accomplish this is to keep the milk at temperatures not exceeding 10° C. (50° F.). The most dangerous temperatures range between 20° C. and 60° C. (68° and 140° F.).

A few years ago it was thought necessary to boil the milk one-half to three-quarters of an hour, but more recent experiments have shown that a shorter boiling has nearly the same effect, while the milk undergoes fewer changes. For instance, the writer, according to the cleanliness of the milk, recommends a boiling of from two to five minutes in winter time and of from five to ten minutes in summer time. The boiling causes certain changes in the milk, which are described in detail on page 311. The most important are a change or coagulation of the proteids, a destruction of the alexines and ferments, change of the salts, etc. It is easy to conceive that these changes are not without importance in the suitability of the milk as food for infants. It is nearly certain that the sterilization of the milk is of influence in the causation of infantile scurvy. Occasionally, delicate infants begin to thrive when they receive raw milk, while previously under otherwise equal conditions the feeding with boiled milk had not been successful. Parallel

experiments conducted with mother's milk have shown that the infants did better on raw milk than on boiled milk (Moro). We may mention here the severe intestinal and fatal disease observed in newborn calves which are raised on sterilized cow's milk, while calves raised on raw milk do not suffer from it. At present, certain general disturbances observed in infants raised on sterilized milk are attributed to the sterilization of the milk. The assumption that the native properties of the milk are lost in the boiling may lend a certain degree of probability to this idea.

Heating the milk for a longer time to 65° to 75° C. (149° to 167° F.) has a similar effect on the bacteria as has boiling. The pathogenic germs are destroyed, while the changes of the milk are less profound. This method of pasteurization has been repeatedly and warmly recommended of late, and special pieces of apparatus have been designed to enable the pasteurization at home. There is a danger that all parts of the milk do not reach the necessary temperature. If only a small quantity remains below this, as may easily happen if a skin is formed, the bacteria are not destroyed, and again infect the rest of the milk in a short time. Special precautions have to be taken to avoid such occurrences, as, for instance, the use of stirring or shaking devices. If the temperature exceeds a certain limit the desired advantages are lost. Special care is necessary in keeping and using pasteurized milk. The less dangerous bacteria can easily be recognized by the changes they produce in the taste and odor of the milk, but the much more dangerous peptonizing bacteria which survive can only be detected with much greater difficulty. Occasionally it may be of advantage to combine sterilization and pasteurization. The milk is kept boiling for two to three minutes and is then placed for fifteen minutes on the hot oven. Thus the milk is gradually cooled to 60° to 70° C. (140° to 158° F.). Then it has to be cooled to 10° to 15° C. (50° to 59° F.) as rapidly as possible.

The addition of disinfecting substances has been tried, in order to render the milk as free from germs as possible without essentially altering its properties. Thus, Behring again recommended the addition of formalin which had formerly been used for this purpose. The addition of preservatives in sufficient dilution may not be of danger to the infant, aside from other serious objections against their use in milk. Seiffert tried to sterilize the milk by exposure to ultraviolet rays, but experiments on a larger scale for practical purposes have not been conducted. With reference to other experiments see p. 328.

Moderate boiling is to be regarded as the surest, most convenient, and cheapest method of freeing the milk from bacteria or reducing their number, without altering the milk too much. Such a procedure is necessitated by the manner in which the milk is obtained. It could be avoided by putting the infants directly to the udder of the animals furnishing

the milk. This method would have the additional advantage that the infants would be forced to do some work in suckling.

Even where good cow's milk was used and the food was prepared rationally, the results never were as good as with breast-milk. Biedert explained this fact as due to the different proteid content of these two kinds of milk. He claimed that the cow's casein and therefore the cow's milk is less digestible than is human milk. This assumption, that the cow's casein is more difficult to digest, has been accepted widely. But more recently its fallacy has been proved, particularly through the investigations of Heubner and the school of Breslau, inasmuch as it has been shown that the resorption of cow's casein is not inferior to that of human casein. But the possibility is not excluded that the cow's milk proteid may act in an injurious manner on the intestines. A new explanation for this possibility came forward recently, to which Biedert calls attention. The biochemical investigations have shown that the proteids of the cow's milk are heterologous for the infant, while those of the human milk are homologous (heterologous, or foreign to the species; homologous, or not foreign to the species). Hamburger drew far-reaching conclusions from this, as it is known that each organism strives to preserve strictly the peculiarities of its species with regard to its cells and body juices, since it reacts against a proteid of a foreign species introduced into its tissues as against a poison. It is the function of the intestines to supply the organism with homologous proteid, and this function is accomplished through the decomposition of the heterologous proteid,—the digestion,—and then through the reconstruction,—the assimilation. These processes may be regarded to a certain extent as a kind of detoxification. The heterologous proteid constitutes the physiological pabulum for the digestive cells in the adult, but it acts as an injury to those of the newborn infant. In contradistinction to the adult, the detoxification of heterologous proteid is not a physiological function of the infant, and in this manner Hamburger explains the injurious effect of cow's milk. Escherich attributes the difficulty of artificial feeding partially to the quantitatively insufficient power of assimilation with regard to cow's proteid. At present we cannot judge with sufficient exactness how far these conceptions agree with the actual facts.

Aside from the high proteid in the cow's milk, the overfeeding of artificially fed babies leads to a further increased introduction of proteid. The digestion of proteids is more difficult than is that of carbohydrates or fats. Thus, we have an expenditure of 10 calories to digest 100 Gm. human milk, and of about 20 calories for 100 Gm. cow's milk. These calories may be utilized by the body for heating purposes, but when the heat excretion is deficient, or when much heat is produced in the body, they may become a burden and require special work for their

removal. The infant, carefully guarded against chilling, may easily be inconvenienced through an excessive production of heat, and a moderate overfeeding even with human milk is therefore not desirable. Overfeeding with cow's milk produces digestive disturbances, with increased peristalsis, increased formation of feces, and gas in the intestines. As a result, the infants cry and become restless, and this again leads to an increased production of heat. The excess of heat leaves the body mainly by evaporation of water through the skin and lungs, as Heubner and Rubner observed in their metabolism experiment on an artificially fed infant.

It is possible that an infant may be raised on whole milk. The greatest care must be exercised in carrying out this method of feeding, and the demand of the infant for excessive amounts of food must be refused with firmness. The amount of milk to be given should be smaller than the corresponding amount in the breast-fed infant. The method is not advisable in delicate and premature infants. It is better to dilute the milk somewhat, at least in the first weeks, but even if these precautions are taken, the infants frequently do not thrive as well as desired. For this reason, the great majority of pediatricians, particularly in Germany, do not adopt this method, but aim to reduce the proteid content of the milk by suitable dilution.

During the first weeks the degree of dilution should be such that the amount of proteid reaches approximately that of human milk. This is accomplished by adding two parts diluent to one part milk. Many people used to dilute the milk still more and even now higher dilutions are sometimes recommended. But it is not rational to employ higher dilutions, for not only are the proteids reduced, but at the same time numerous other substances are diminished which the infant needs. In particular, the caloric value of the food decreases to such an extent that the necessity for a considerable increase of the total volume can hardly be avoided, in spite of the addition of suitable constituents. The stomach and the intestines are thus directly burdened, and the total metabolism is overcharged (increased work of the heart, vessels, and kidneys, increased production of sweat, with its consequences). As to whether infants recovering from disorders of the nutrition should not receive higher dilutions for a short time is another question.

The time of transition to more concentrated milk mixtures is given differently by different writers. The French physicians, for instance, give half-diluted milk after a fortnight, while in Germany this dilution is rarely given before the end of the second month. It will be best to be guided in a given case by the state of the infant's health and particularly by its digestive power. Sometimes the necessity arises to give general advice. If an infant is healthy, we very cautiously try to give half-diluted milk in the third or fourth week, and increase to two parts

of milk to one part diluent in the eighth week, three parts milk to one part diluent in the fifth month, and whole milk in the eighth month. With regard to the nutrition after the eighth month to the end of the first year, see p. 393. Sometimes it will be necessary to give the stated dilutions for a longer period of time or to return to a higher dilution. The transition should be gradual, so that at one to two day intervals one bottle of the more diluted solution should be replaced by one of greater concentration.

Cereal decoctions or water are mostly used as diluents. Steffen recommended thin meat broths, and Monti whey. Since these diluents are wholly or to the greatest extent composed of water, an undesired diminution of the carbohydrates and fat takes place with the desired reduction of the proteids, and thus the nutritive value is markedly impaired. This disadvantage cannot be corrected by a corresponding increase in the amount of food, and an addition of one or more food materials is required.

Fats and carbohydrates may be added. The use of both seems to be the most rational, since thus we approach most nearly the natural conditions, and as a matter of fact such mixtures are widely employed.

Biedert's natural cream mixture and the cream conserve (Ramogen) should be mentioned. The former is prepared by mixing cream, water, and sugar in the following manner:

TABLE 20.

1	2	3	4	5	6	7	8	9	10	11
Mixture.	Cream, part.	Water, parts.	Milk, parts.	Sugar to 100 water.	Sum of the parts.	Each part about c.c.	Proteid, Gm.	Fat, Gm. in 100 c.c.	Sugar, Gm. in 100 c.c.	Calories.
I	1	3		(5) 6 per cent.	4	25- 50-100	9	25	(46) 56	(460) 500
II	1	3	$\frac{1}{2}$	(5) 6 per cent.	$4\frac{1}{2}$	50-100-200	12	26	(46) 55	(475) 515
III	1	3	1	(5) 6 per cent.	5	100-200	14	27	54	585
IV	1	3	2	(5) 6 per cent.	6	100-200	17	29	52	550
V	1	2	4	(5) 6 per cent.	7	150-200	25	35	50	690

The cream is figured at 10 per cent. fat, 3.6 per cent. proteid and 4.5 per cent. sugar. Mixture I is to be used in the first month, II in the second, III in the third to fourth, IV in the fourth to fifth, and V in the sixth to seventh. Since the fat content of cream, which is not obtained by the use of machinery, varies considerably and as it is difficult to obtain suitable cream particularly in summer time Biedert caused the above mentioned cream conserve to be prepared. It is put on the market in tin cans containing 260 Gm. and contains about 7 per cent. proteid, 15 per cent. fat, and 35 per cent. sugar (about 10 Gm. of this are milk-sugar and 25 Gm. cane sugar). The cream conserve is given diluted with water or milk as desired. Biedert recommends the following mixtures:

A. RAMOGEN WATER EMULSIONS.

1. Ramogen 1 : 6 water, equal to 1.0 per cent. proteid, 2.2 per cent. fat, 5.1 per cent. sugar; 450 calories in 1000 c.c.

2. Ramogen 1 : 5 water, equal to 1.2 per cent. proteid, 2.6 per cent. fat, 5.9 per cent. sugar; 590 calories in 1000 c.c.
3. Ramogen 1 : 4 water, equal to 1.4 per cent. proteid, 3.2 per cent. fat, 7.1 per cent. sugar; 650 calories in 1000 c.c.

B. RAMOGEN MILK EMULSIONS.

1. Ramogen 50 Gm., milk 125 c.c., water 575 c.c., sugar 20 Gm., equal to 1 per cent. proteid, 1.75 per cent. fat, 6 per cent. sugar; 450 calories.
2. Ramogen 100 Gm., milk 250 c.c., water 650 c.c., sugar 10 Gm., equal to 1.64 per cent. proteid, 2.55 per cent. fat, 6.2 per cent. sugar; 550 calories.
3. Ramogen 100 Gm., milk 500 c.c., water 400 c.c., sugar 30 Gm., equal to 2.25 per cent. proteid, 3.5 per cent. fat, 6.5 per cent. sugar; 680 calories.

The composition of all preparations of infants' foods should be thoroughly understood. Only in this way is it possible to use them rationally and at the same time to exercise some control. It would be desirable if physicians would reject all preparations which do not satisfy this demand.

The numerous preparations, some of which have been in use for many years, show that the method of adding cream with or without the addition of carbohydrates has given good practical results. The particular method and the preparation which should be used in a given case depends upon circumstances. As it is the use of fresh cream would be preferable. But only with difficulty can this be obtained to meet all requirements, particularly in summer time. The general objections against the use of proprietary foods are not so great when we take into consideration the dangers of an unsuitable cream, and furthermore the composition of the preparations is much more constant and their use simpler. A prolonged use of these preparations, however, leads to certain dangers. Many infants do well on the fat preparations, but a considerable number do not tolerate an increased amount of fat, and react sooner or later with intestinal disturbances. The increased ingestion of fat may increase the acidosis, with its detrimental effect on the total metabolism. Caution should therefore be exercised in increasing the fat in the infant's food and the deficiency of the diluted milk in calories should not be corrected by the addition of fat alone.

The second group of additional food materials is composed of carbohydrates. Disaccharides and polysaccharides enter into consideration, while monosaccharides are hardly ever used. It was natural to use the milk-sugar, a disaccharide, particularly since its recommendation by Soxhlet, Heubner and Hofmann; it is employed with success in rearing numerous infants. But it soon became manifest that the more concentrated solutions of milk-sugar caused diarrhoea, and the gain in weight did not always correspond to the amounts of milk-sugar given. These two phenomena may be partially due to the fact that certain amounts of lactose are decomposed in the intestines through the action of bacteria and are thus lost to the energy metabolism. It may be best to add from about 5 to 7 per cent. milk-sugar during the first weeks of life.

The use of cane sugar is somewhat limited, as it causes fermentation and diarrhœa in the higher concentrations. Its sweetness may lead to repulsion or may cause a refusal of other food. However, if the infant is older, an addition of from 2 to 5 per cent. cane sugar in combination with other nutritive substances may be regarded as suitable.

The last disaccharide to be mentioned is maltose. It is not used in a pure state, but in combination with other substances as with dextrin. Soxhlet's "Nährzucker" contains both these substances, in about equal parts; improved Liebig soup containing about 60 per cent. maltose and 20 per cent. dextrin; Keller's malt soup contains in 1000 Gm. soup, 100 Gm. malt soup extract, and 50 Gm. wheat flour; Mellin's food contains about 50 per cent. maltose and 35 per cent. dextrin. The use of maltose seems to have been first introduced into pediatrics by Liebig in the form of his malt soup.

The use of the polysaccharides, flour and dextrin, has fallen into discredit. The unfavorable results of feeding with gruels is responsible for this, beside the opinion that the diastatic power of the salivary glands and of the pancreas is not sufficiently developed in the young infant. Thus, many deem it a mistake to add flour before the tenth month. Recent investigations have revealed the fact that small amounts of flour can readily be digested so early as the first weeks of life, and that it is frequently possible to obtain very good results even at this time, but particularly so later on, after cautious additions of flour or dextrin to the food. The influence on the movements of the bowels is frequently very favorable. Constipation is relieved, the fat and soap stools disappear, and the passages become uniform and soft. Nevertheless, the young infant should be watched carefully when fed on flour, since an excess may occasionally cause sudden catastrophes. As soon as the passages give a distinct reaction for starch, or become very acid, the addition of flour has to be reduced or stopped entirely. As a rule, such preparations may be added to the food mixture in the following amounts: during the first month about 1 per cent., during the second 2 per cent., during the third 3 per cent., and from then on 4 to 5 per cent.

The dextrinized flours are to be recommended, not so much perhaps on account of practical observations as on the basis of theoretical considerations. They always contain more or less starch beside the dextrin. Their main representatives are the flours for infants, some of which are prepared with an addition of sugar, and, rather irrationally, with milk (as, for instance, that of Nestle). The table on the following page informs us of their composition.

The percentage of soluble carbohydrates varies greatly. The manufacturers like to use this fact as a basis for their statements, frequently in a very objectionable manner. Zwieback, which is cheap, is very useful for children, and contains much less dextrin.

TABLE 21.

	Proteid, per cent.	Fat, per cent.	Carbohydrates.		Water, per cent.
			Soluble.	Insoluble.	
Teinhardt, soluble infants' food.....	16.1	5	53.6	16.7	5
Nestle, flour for infants.....	9.9	4.5	42.5	35.2	6
Muffler, food for infants.....	14.2	5.5	29.0	43	5.6
Kufcke, flour for infants.....	13.6	1.8	23.5	51	8

Furthermore, we have at our disposal preparations of fine flour derived from oats, rice, or corn. These are manufactured in good quality and at very reasonable prices. They contain about 60 to 70 per cent. insoluble and 2 to 5 per cent. soluble carbohydrates. Small amounts (1 to 5 per cent.) of insoluble starch and to a small extent of soluble starch are to be found in the cereal decoctions (from oat, rice, and barley meal), which are frequently used as diluents, particularly during the first week. These are prepared as follows: One tablespoonful of the meal is treated with 1 pint of water and a little table salt, the mixture is boiled half or three-quarters of an hour, and is then strained; if desired, the evaporated water is replaced.

A third group comprises foods prepared from cow's milk and made as similar to human milk as possible. To accomplish this humanization, the required amounts of cream and milk-sugar are added to the diluted milk (Gärtner's milk, see p. 327), or whey is used instead of water (Monti, see p. 327; former Backhaus milk; lait humanisé of Winter-Vigier). Others remove part of the casein by means of carbon dioxide (Szekely milk, see p. 327); and others predigest the milk proteid (Volmer-Lahrmann; Backhaus, Baumann, see p. 328). Then, again, the casein is replaced by egg albumin (Hesse-Pfund), or by an albumose obtained from chicken proteid (Rieth), or by somatose (somatose-ramogen and milk-somatose-ramogen). In other instances, a proteolytic enzyme is added to the mixture (milk powder Timpe, pankreon, papain, pepsin). The pepsin milk (obtained, according to von Bungern, through addition of rennin) is supposed to render the casein of the milk more easily digestible on account of its very fine coagulation. Experience has taught us that we may be able to raise an infant on any one of these more or less changed milks. But it is doubtful whether their use offers any advantage. Even the theoretical reasons which prompted some of the different methods, have in the course of time been proven to be fallacious. It certainly is essential that the milk used as the basis for the different preparations be beyond reproach. No doubt the treatment of the milk, and particularly long-continued boiling, produces marked changes in it, and the prolonged use of such milk may be followed by serious disturbance of the metabolism. Furthermore, the preparations are often not consumed when still fresh, but are stored

eight or even fourteen days. If not kept under the proper conditions, processes of decomposition may be started which are the more dangerous in that they do not betray themselves immediately to the eye, nose, or taste. A further disadvantage is the high price of most of the preparations. In spite of all this, it may happen that a previously poorly nourished infant improves rapidly after starting it for instance on Backhaus or Gärtner milk. This is hardly to be attributed to the method as such, however, but to the fact that good milk is used and that the food is ready for drinking, so that the persons entrusted with the care of the infants have no opportunity to spoil anything.

In Mehring's Odda the milk fat is replaced by cocoa butter and yolk of egg. The cocoa butter does not become rancid. There have yet not been sufficient observations to decide the value of this preparation.

We will briefly call attention to buttermilk, which is mostly used for sick infants in Germany, but in Holland healthy infants are raised on it with the best results. Its composition is given in detail on p. 329. To make it ready for use, 10 to 15 Gm. wheat flour and 60 to 70 Gm. cane sugar, or a desired amount of cream are added to one litre buttermilk; the whole mixture is boiled slowly about half an hour, with stirring. If good buttermilk (the usual product of the market is unfit for use) is not to be had, a buttermilk conserve may be used ("ferment milk" and lactoserve). Further investigations must be made to determine whether buttermilk can be recommended as a constant diet for the healthy infant.

TECHNIQUE OF ARTIFICIAL FEEDING

After considering the different methods of artificial infant feeding, its technique and practical application will be discussed. The age, weight, and state of health of the infant serve as guides in the formation of the plans for feeding. These factors determine the quality and number of meals. Weight and age furnish us information as to the amount of energy to be supplied. During the first four months, as previously explained, about 110 calories have to be introduced per kilo, during the second 100, and during the third 90. In order to simplify the calculations, the calorimetric values of the most important infants' foods are given, according to Salge, in the table on the following page.

These figures cannot be regarded as absolutely correct; as, for instance, the calorimetric value of cow's milk varies between 500 and 700 calories, according to its lower or higher content of fat, etc. If under normal conditions the energy quotient deviates for a long time from the figures stated above, disturbances are to be expected. Furthermore, the weight and age of the infant determine the absolute and relative proteid content of the food or the milk concentration, and also the amount at each feeding and for the day. Finally, the economic condi-

tions of the family are of importance, since the more expensive preparations and selected milk properly delivered cannot be used; moreover, the degree of intelligence is to be considered, as well as the interest of the persons who are intrusted with the carrying out of the feeding.

TABLE 22.
Calorimetric values of the most important infants' foods.

	100 c.c.	200 c.c.	300 c.c.	400 c.c.	500 c.c.	600 c.c.	700 c.c.	800 c.c.	900 c.c.
Human milk	70	140	210	280	350	420	490	560	630
1/2 milk with 5 per cent. decoction of infants' flour and addition of milk-sugar according to Heubner	40	80	120	160	200	240	280	320	360
1/2 milk, otherwise same	46	92	138	184	230	276	322	368	414
1/2 milk, otherwise same	52.5	105	157.5	210	262.5	315	367.5	420	472.5
1/2 milk and 8 per cent. Soxhlet Nähr-sucker	54	108	162	216	270	324	378	432	486
1/2 milk, otherwise same	67	134	201	268	335	402	469	536	603
1/2 milk, otherwise same	78	156	234	312	390	468	546	624	702
Buttermilk	71.4	142.8	214.2	285.6	357	428.4	499.8	571.2	642.6
Malt soup, Liebig-Keller	80	160	240	320	400	480	560	640	720

Sometimes we may adhere with advantage to a certain fixed scheme of feeding, but such a procedure does not enable the individual case to be carefully considered. Biedert elaborated such a scheme and used the percentage method, and Escherich advanced another based on the volumetric method. Such schemes may be very different, depending on the social condition, intelligence, customs and habits of the region, and the material at one's disposal (for instance, the milk of mountainous regions being rich in fat, and that of the plains poor in fat). The following scheme proved successful as a rule in the middle classes of Southern Germany:

TABLE 23.

	No. of meals.	Total amount of		
		Milk (litre.)	Dilute (litre.)	Addition (for instance, milk-sugar, cane sugar, etc.) in grams.
1st week	7	$\frac{1}{8}$	$\frac{1}{8}$	25
2d to 3d week	7	$\frac{2}{8}$	$\frac{2}{8}$	45
4th to 8th week	7	$\frac{3}{8}$	$\frac{3}{8}$	45
3d month	6	$\frac{4}{8}$	$\frac{4}{8}$	40
4th month	6	$\frac{5}{8}$	$\frac{5}{8}$	40
5th to 6th month	6	$\frac{6}{8}$	$\frac{6}{8}$	30

The transition is to be accomplished very gradually, so that one bottle of the more diluted food is replaced by one of the more concentrated. If the infants are very robust, we proceed more rapidly than is indicated in the table.

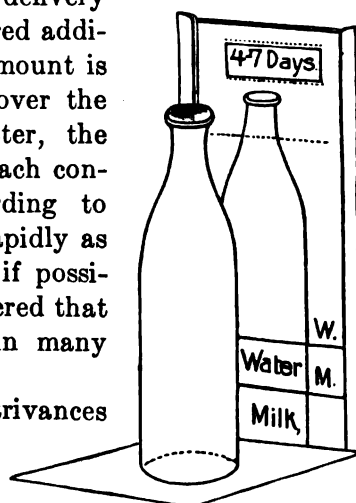
Since the food for the infant is frequently prepared in a faulty manner in spite of all directions, and since people not accustomed to it find great difficulties in measuring out required quantities of milk, the author constructed a model for mixing, which obviates these difficulties. It is a stand made of sheet iron, and several cards are provided which can be displayed on the vertical part of the stand. Each card shows the

picture of a bottle with marks fixed at different heights (according to the different ages of the infants) which indicate the amount of milk, etc., to be put in the actual bottle standing opposite the picture.

The following may be added to what has been said with regard to the handling of the milk at home: The milk should be mixed and strained immediately after its delivery and then diluted as desired, and the required additions be made. Then either the total amount is boiled (in so-called milk boilers (Fig. 54) over the free flame or in the water-bath), or, better, the milk is first distributed into the bottles, each containing the amount for one meal (according to Soxhlet). The cooling must take place as rapidly as possible after the boiling, in summer time if possible in the refrigerator (it must be remembered that the temperature remains relatively high in many refrigerators), or in running water.

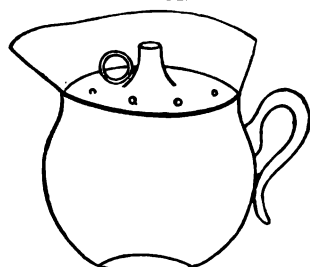
The bottles are closed with the contrivances introduced by Soxhlet, Ollendorf, and Stutzer. These are expensive and do not last long. The stoppers of Raupert (Fig. 55) are cheap and easily cleaned, and are therefore to be recommended. The bottles should be smooth on the inside and the corners of the bottom should be rounded. If possible the bottles

FIG. 53.



Milk modifying device (Camerer).

FIG. 54.



Flüge's milk boiler.

FIG. 55.



Bottle stopper (Raupert).

should be made of hard glass. [In the United States, cylindrical bottles with rounded interior are readily obtained; non-absorbent cotton makes the best stopper.]

Simple nipples are used or those which resemble the natural nipple. ["Hygeia" nipples.] Both kinds are cheap and easily cleaned. By changing the size of the hole in the nipple, the outflow and consequently the work of suckling may be regulated to a certain extent. Nipples

provided with a long tube should be absolutely forbidden, as they cannot be thoroughly cleaned.

Materials composed of rubber or hard rubber must be cleaned immediately after use. They are rinsed out with warm water, and the inside and outside are rubbed with salt and again rinsed with water, and occasionally they are boiled; they are kept in clean well covered vessels. The bottles should be cleaned with water immediately after use. Each bottle must be cleaned daily with a hot solution of sodium carbonate and a brush, and then be rinsed with water. They should be kept dry, standing bottom up. If held against the light, the bottles should not show any cloudiness.

CHILDREN'S GROWTH IN WEIGHT AND HEIGHT

BY

DR. W. CAMERER, OF STUTTGART

TRANSLATED BY

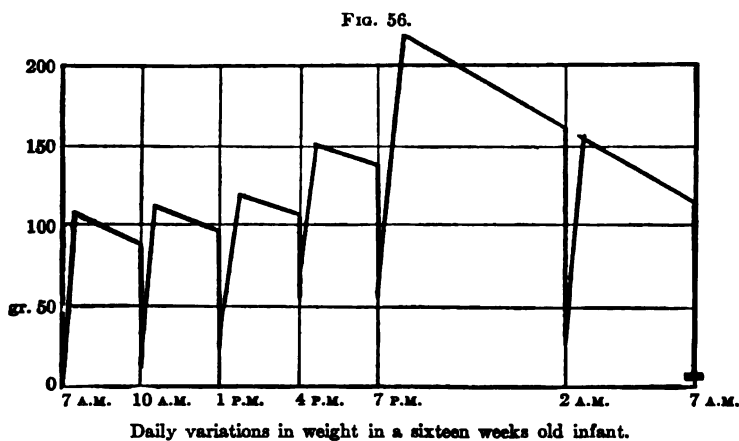
DR. SAMUEL AMBERG, BALTIMORE, MD.

ONE of the most interesting tasks of anthropology is to investigate the growth of children from the time of birth to the completion of the period of development. The physician and hygienist must possess a knowledge of the processes of growth. This knowledge enables him to judge whether and how far the growth of a given individual deviates from the normal, and furnishes him the indication for his therapeutics. The hygienist has for his field the prevention of disease, and he must be well informed about the processes of growth in order to recognize and combat intelligently the many dangers to which the growing organism is exposed and the injuries which it so frequently sustains. This is particularly true for the school hygienist. There is a close relation between growth and the schools. We may here call attention to the influence which the length of the children has on the shape and construction of the school benches, and also on the condition of the school room, the division of the school hours during the day, the duration and timing of the recesses (particularly of the noon-day recess), the interpolation of physical exercises between the school hours proper, and the duration and the season of vacations.

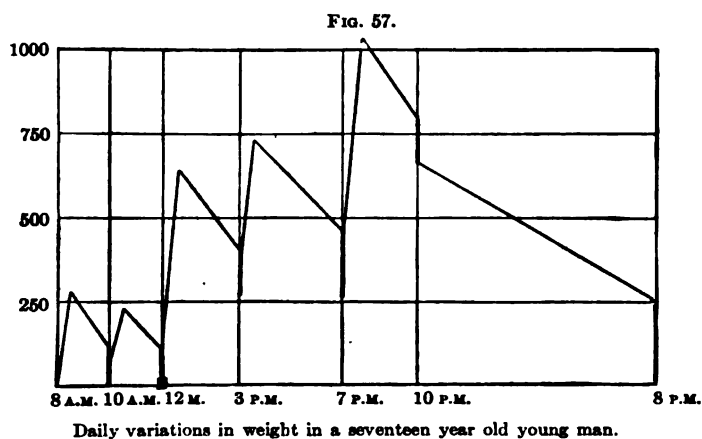
Growth in its more restricted sense means those processes in the healthy youthful body which, following the laws of evolution, lead to an increase in size, weight, and mass of the total body and its individual parts.

Investigations with regard to the growth of single organs, as for instance the brain, or of single systems, as the muscles, are extremely difficult, and our knowledge is rather incomplete in this regard. It is more simple to study the growth of the body in regard to its weight and length. For instance, the opinion is frequently voiced that it is possible to obtain satisfactory information of the growth of an infant if it is weighed at certain intervals and its length is determined, the comparison of the data thus gained furnishing the desired information. It can be easily shown that such a procedure may lead to great mistakes. For, while the growth forms the most important cause of the increase in weight and length of an infant, it is by no means the only one. Numerous other factors may change the weight, and to a less degree the length, either increasing or diminishing it. The variations of the weight in the

course of 24 hours are quite considerable. The lowest weight is registered in the morning before breakfast, the highest in the evening after supper. The difference between the morning and the evening weight is about 200 Gm. in the case of an infant four months old, about 700 Gm. in a child ten years old, and about 1000 Gm. in the adult. This increase in weight is due to the fact that the intake exceeds the excretions during



the day. The loss of weight from evening to morning is chiefly caused by the elimination of water through the kidneys, skin, and lungs during the night (Fig. 56). This loss of weight is, on an average, equal to the gain in the case of the adult. In the growing child it is somewhat less than the gain during the day (Fig. 57). The increase of weight from



morning to evening does not progress at a regular rate, but is subject to great oscillations, corresponding to the continuous change of the external conditions—ingestion of food, excretions, temperature, humidity of the air, occupation. The decrease of weight during the night proceeds more regularly. This may best be seen on the curves of Figs. 56

and 57 (observations of the elder Camerer). In order to explain Curve I, the following may be added: An infant 16 weeks of age receiving mother's milk weighs 5200 Gm., and this weight is registered in the figure as O. From 7 to 7.30 A.M. it took 107 Gm. mother's milk (represented by the ascending line) and the weight increased 107 Gm. Between 7 and 10 A.M. it lost 28 Gm. in the form of gaseous excretion (represented by the second more horizontal line) and secreted 64 Gm. urine (represented by the vertically descending line). In consequence of these processes the weight at 10 o'clock exceeds that of 7 o'clock before nursing by only 15 Gm. The nursing at 10 o'clock increased the weight 122 Gm. above the initial weight, etc. The figures of Curve II were obtained in a youth seventeen years of age. The body weight of 52800 Gm. is registered as O. The increase in weight is due to the intake of food; the decrease, to excretions through the intestines, kidneys, skin and lungs. The broad line placed in Fig. 56 at 7 o'clock A.M. and in Fig. 57 at 12 noon designates defecation.

Insufficient or excessive feeding has a considerable influence on the body weight; for instance, if poor city children enter fresh air colonies, their weight increases considerably during the few weeks they live in the country and receive an abundant supply of food, but frequently they lose this gain after returning to their former life. Manifestly such a gain is more a matter of fattening them than an expression of growth. Frequently we find such an overnutrition in cases of sucklings, many of whom do not progress very well with their general development in spite of their great weight, and who lose their excessive weight when, for instance, they pass from the excessive milk diet to the table food.

The length is also subject to certain variations, but these cannot be demonstrated as easily as can the variations of the weight. Children and adults are 1 to 3 cm. longer immediately after the night's rest in bed, than a few hours later. In case of great fatigue the length of the body may be shortened as much as 4 to 5 cm.

If we consider that for several years the total yearly increase in length is only 5 cm., measurements disregarding these variations may lead to very erroneous results. The daily decrease in length does not proceed regularly during the day, but is subject to continual variations. The decrease begins immediately after rising and reaches its maximum about four to five hours later. After lying for some time in a horizontal position (in the afternoon, for instance), the body regains its maximal length. The variations are mainly caused by the following circumstances: The erect position leads to a compression of the cartilaginous layers between the single vertebræ, the spinal column becomes curved, and the vault of the foot becomes depressed. A more pronounced erect position increases the length a little, and most recruits are therefore a little longer after a short time of service. But all this has nothing to do with

growth in a more restricted sense, just as little as has the increase of length in the case of children who have been kept in bed on account of disease.

The exact determination of the length is difficult in itself, and, particularly in the case of infants, is very much more complicated than the determination of the weight. An excellent observer, the late Professor Wiener, found differences amounting to 3 mm., measuring repeatedly within short intervals; observers of less skill will easily make errors of from 0.5 to 0.7 cm., even if their method is good.

Beside the daily variations of weight and length, there are regular variations in the course of the seasons, and these are not connected in any way with the growth. The greatest gain in weight in the course of the year occurs in the Fall, from August to December; the smallest gain is registered during the Spring, from April to July. The increase in length is exactly reversed.

Many factors, therefore, have to be considered if we want to determine the weight and length of the body, and to use the results obtained for the determination of the growth. The weighing should be done in the morning before any food is given, and without any clothes, or with as little clothing as possible, and the length should be measured in the morning immediately after leaving the bed.

It is desirable to obtain reliable average values from observations on a larger number of children, since the weighing and measuring of a given case may be subject to accidental mistakes. Such values are indispensable for the physician and hygienist. Two different methods may be employed in order to gain these average values, the generalizing, or method of collective investigation, and the individualizing. Using the first method, numerous children of about the same age (for instance, members of a class in school) are weighed and measured to form an average value for a given age. In this way it is possible to obtain values of weight and length for the total period of growth within a short time. It can readily be understood that this method cannot give satisfactory results if the material of observation is not absolutely uniform. Age and sex, the race, social condition, state of health, time of year and day, etc., must be taken into consideration most carefully. Using the second method, the weight and length of single individuals are determined during the total period of development, and by combining numerous such observations in a suitable manner we arrive at average values. This laborious and tedious method must be adopted if we want to gain information about the finer processes of growth. While the collective investigation reduces or annuls the influence of accidental variations, it effaces at the same time regular variations and influences, the knowledge of which is desirable and necessary. For instance, during the first year of life the expected influence of teething and weaning can only be

elicited from observations on the single individual. By weighing several infants five months old, others six months old, and again others seven months old, we can obtain the average weight for the given age. But the influence of teething and weaning is entirely effaced, because these events do not occur in all these infants at the same age, but in one case in the fifth, in the other in the sixth month. Using the generalizing method, we can make certain subdivisions in order to study the effect of special influences, those of wealth and of poverty, for instance. But the subdivision is made here before the observations are taken and according to a preconceived idea. Using the individualizing method, we can make suitable subdivisions on the basis of the results obtained by observations on single individuals, remembering that these results will be conditioned by the degree of uniformity in the material. Therefore, this latter method furnishes the best results, since it enables us to observe the growth of the individual and to arrive at suitable average values.

Quetelet weighed ten individuals of male and female sex of all periods of age during the total time of growth. Naturally, these observations are insufficient in many regards, and it is surprising that even to-day they are used exclusively in many statistics. Quetelet had many followers, among whom, Bowditch for instance, made a very careful statistical study of growth; his observations were made on several hundred individuals classified according to age. Very few continuous observations of the growth of individual children were at our disposal until about 25 years ago. Since then Camerer (the elder) has collected and worked out a rich material of very carefully observed individual cases, and at present several hundred cases of the first years of life are at our service. The figures given in the following are chiefly based on these observations.

The birth weight of normal healthy German boys of the middle classes is about 3400 Gm., that of girls 3200 Gm. The sex of the infants, the social condition and race of the parents, the term of birth, the number of previous births, and other factors, exercise a considerable influence on the birth weight and are not taken sufficiently into consideration. For instance, the average birth weight is often given as 3000 Gm. The reason for this is that the first and most frequent investigations with regard to the birth weight were conducted in maternity hospitals. Nearly all the mothers admitted to such institutions live in poor circumstances and frequently do hard physical work until a short time before parturition. According to the data of Fehling, the birth weight of children of women working in factories is the lowest, then follow those of servant girls, seamstresses, and shop girls. The birth weight of such infants is relatively low on account of peculiar conditions, and cannot be taken as normal.

The injuries sustained by the infants during parturition cause a loss of weight of about 200 Gm. during the first days of life. The first day participates in this loss with about two-thirds, the second with one-third. But even from the first day on, a small gain of weight is noted in most cases, and the infants regain their birth weight on the eighth to the tenth day. From then on the gain in weight proceeds as follows:

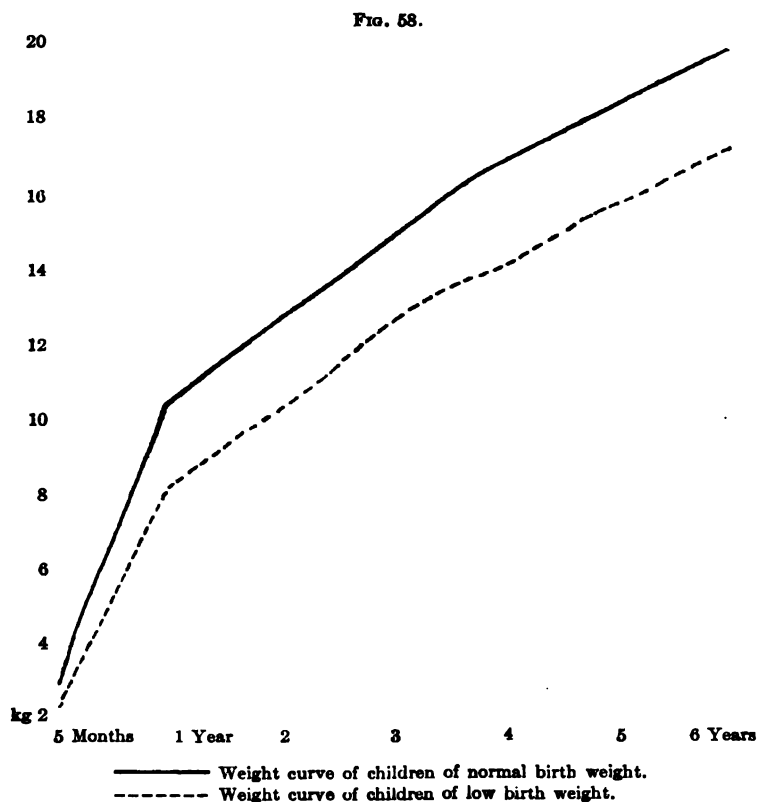
TABLE 1.

	Breast-fed infants.		Artificially fed infants, male and female.
	Male.	Female.	
Birth weight	3480 Gm.	3240 Gm.	3390 Gm.
End of 4th week	4170 Gm.	3810 Gm.	3730 Gm.
End of 8th week	5080 Gm.	4560 Gm.	4340 Gm.
End of 12th week	5870 Gm.	5270 Gm.	4950 Gm.
End of 16th week	6580 Gm.	5900 Gm.	5610 Gm.
End of 20th week	7140 Gm.	6520 Gm.	6270 Gm.
End of 24th week	7650 Gm.	6920 Gm.	6900 Gm.
End of 28th week	8140 Gm.	7380 Gm.	7300 Gm.
End of 32nd week	8540 Gm.	7800 Gm.	7750 Gm.
End of 36th week	8900 Gm.	8090 Gm.	8130 Gm.
End of 40th week	9220 Gm.	8400 Gm.	8270 Gm.
End of 44th week	9650 Gm.	8720 Gm.	8650 Gm.
End of 48th week	9970 Gm.	8970 Gm.	8910 Gm.
End of 52nd week	10210 Gm.	9660 Gm.	9980 Gm.

The daily gain of breast-fed infants is about 30 Gm. to the fourth week; 26 to 28 Gm. from the fifth to the twelfth week; 20 to 24 Gm. from the 13th to the 20th week; 16 to 18 Gm. from the 21st to the 36th week; 10 to 15 Gm. from the 37th to the 52nd week. Accordingly, the weight of breast-fed infants is about 4000 Gm. at the end of the first month; at the beginning of the fifth month the weight is about double the birth weight, and at the end of the first year it is about three times the birth weight. The difference in the weight of boys and girls increases gradually more and more in favor of the boys and at the end of the first year it amounts to about 500 Gm., so that at this time the boys weigh about 10.2 kilos, the girls 9.7 kilos.

The course of the growth during the first year can best be shown graphically. The curve of growth shows in the case of breast-fed infants (see Fig. 59, and the explanation, on p. 415) an uninterrupted rise with gradually increasing acceleration, except for the fall in the first days of life, the reasons for which have been discussed. In the third quarter a second insignificant inhibition of growth occurs, with a consequent transitory acceleration; this inhibition is due to the weaning and to the development of the teeth. Artificially fed infants remain considerably behind the breast-fed in the first quarter. In this period their daily growth is only about 22 Gm., but until about the sixth month it keeps itself rather constantly on this level, while the daily growth of the breast-fed infants increases from the fourth week on, as previously pointed out. Irrational feeding and disorders of nutrition are responsible for the small gain of the artificially fed infants during the first quarter. The constancy of the gain in the second quarter is to be regarded as a

manifestation of convalescence. At the end of the first year the artificially fed infants reach about the same weight as the breast-fed ones, provided the feeding has been rational. Therefore, the weight at the end of the first year is not dependent on the kind of food but on the birth weight. This becomes particularly clear in the case of infants with an abnormally small birth weight. Such children may stay behind infants with normal birth weight at the end of the first year and remain considerably behind for many years to come (see Fig. 58); as to whether they were breast-fed or raised artificially makes no difference.



The cause of the gain in weight during the first year of life under the different conditions is best illustrated in Fig. 59. The uppermost, red curve indicates the gain in weight of fifty-seven healthy breast-fed boys under continuous observation; the second, yellow curve, the gain of 114 breast-fed boys and girls; the third, green curve, the gain of 85 artificially fed boys and girls, all of these infants weighing at birth between 3 and 4 kilos; the fourth, black curve, the gain of 24 boys and girls with an average birth weight of 2400 Gm.; the fifth, dotted curve, the gain of fourteen boys and girls with a birth weight below 2 kilos. Furthermore, these observations show that the frequently constructed

"normal curve for the growth in the first year of life" has only a very limited value.

In the second year of life the gain in weight is very much less than in the first. In boys and girls the gain is only about 2.5 kilos and from the third to the fifth year it decreases still more, to 1 to 2 kilos per annum. At the end of the fifth year boys have a weight of about 18 kilos, girls of 17 kilos. From then on the weight of boys increases 2 to 3 kilos per annum to about the 14th year; then follows a period of increased growth, with a yearly gain of about 8 kilos from the fifteenth to the eighteenth year. The yearly weight in girls is about 2 kilos to the twelfth year; it then increases to 4 to 5 kilos from the thirteenth to the sixteenth year. The gain in weight due to the processes of growth is practically completed by the end of the sixteenth year in girls and the nineteenth year in boys. The following table gives in round numbers the yearly growth in weight:

TABLE 2.

Year of life.	Boys.		Girls.	
	Body weight, in kg.	Yearly gain, in kg.	Body weight, in kg.	Yearly gain, in kg.
Birth	3.4	...	3.2	...
1	10.2	6.8	9.7	6.5
2	12.7	2.5	12.2	2.5
3	14.7	2	14.2	2
4	16.5	1.8	15.7	1.5
5	18	1.5	17	1.3
6	20.5	2.5	19	2
7	23	2.5	21	2
8	25	2	23	2
9	27.5	2.5	25	2
10	30	2.5	27	2
11	32.5	2.5	29	2
12	35	2.5	32	3
13	37.5	2.5	37	5
14	41	3.5	43	6
15	45	4	48	5
16	50	5	52	4
17	56	6	...	...
18	60	5	...	...

Growth in weight.

Certainly only few individuals keep exactly the weight reached in the sixteenth to nineteenth year of life. The majority continue to gain weight gradually mostly through retention of fat and increase of the muscles. For this reason, in establishing average values a slow increase in weight is noted until the twenty-fifth year. But this gradual change of the body cannot be designated any more as "growth." Every healthy human being *must* gain in weight up to the sixteenth to nineteenth year of life, but from that time on the weight depends on the individuality and on external accidental influences,—such as, for instance, the occupation and the food. The side curve in Fig. 60 illustrates this very clearly.

This curve illustrates the events as observed in a healthy youth. With seventeen years he had reached a weight of 60 kg. (I); with

seventeen and a half years he rose to 61.8 kg. (II) (Figs. 59 and 60); while preparing for an examination the weight dropped 2.6 kg. (III)

• FIG. 59.

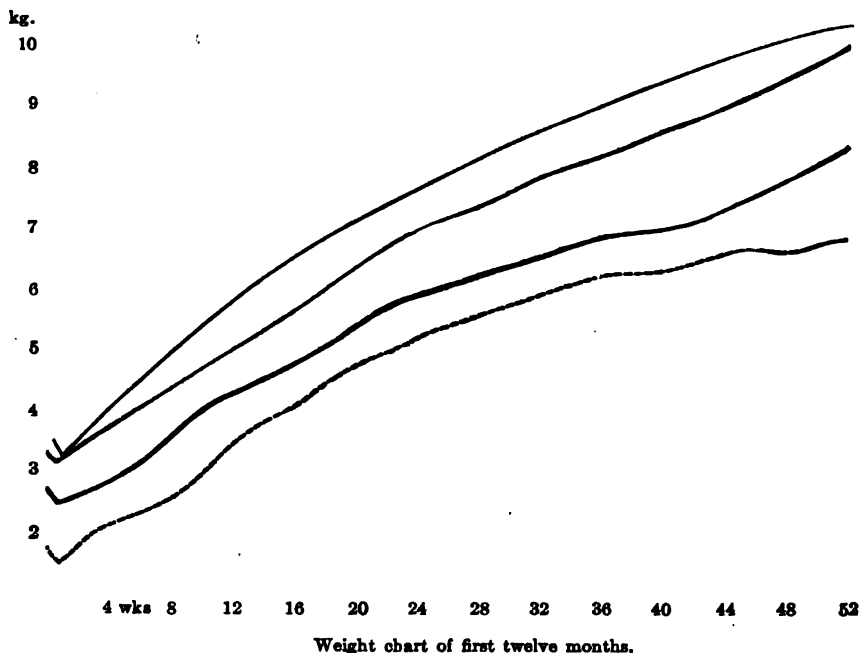
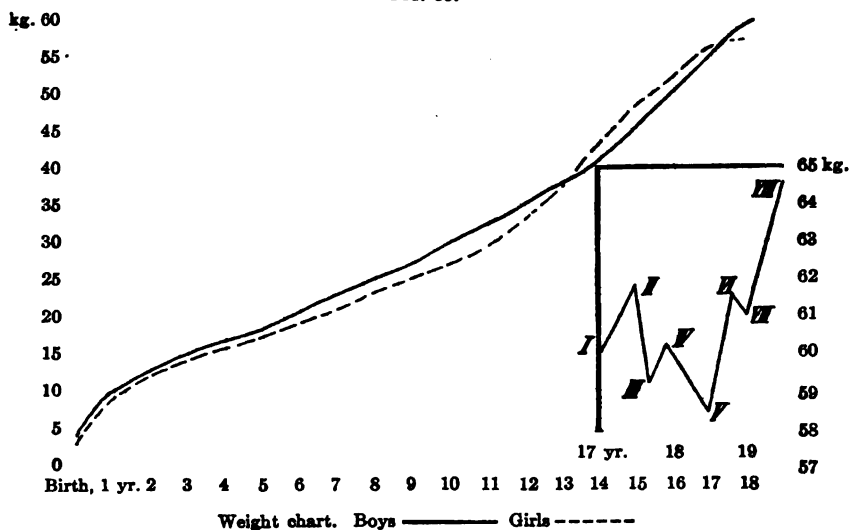


FIG. 60.



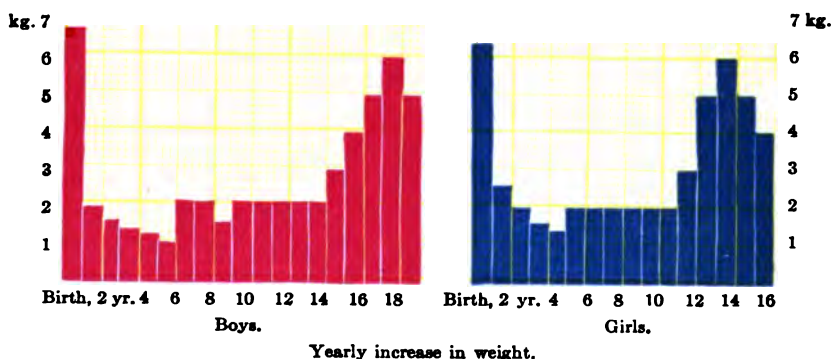
in the course of three months. During two months' vacation the weight increased 800 Gm. (IV), to drop $2\frac{1}{2}$ kg. (V) during the next six months of hard studying. The next half year brought a gain of 3 kg. (VI) while

serving in the army; then a febrile disease rapidly decreased the weight $\frac{1}{2}$ kg. (VII), and in the next half year of lighter study a gain of $3\frac{1}{2}$ kg. (VIII) was noted.

The relative figures of growth are very interesting, aside from the absolute figures. In the first month of life the daily gain in weight is roughly 1 per cent. of the present body substance, in the middle of the first year about 0.3 per cent., and at the end of the first year still 0.15 per cent.; then it reaches a minimum in the fifth year, with about 0.03 per cent., to increase in the second great period of growth to 0.04 per cent. in girls and to 0.07 per cent. in boys. The ingestion of large amounts of food, frequently observed in growing boys from fourteen to eighteen years of age, is not caused by the growth, as is usually supposed, for the daily gain is at this time only a few grams and in relation to the body substance is infinitesimal. The growth therefore cannot play an important rôle with regard to the processes of the metabolism.

Reviewing the total cause of the gain in weight, we can distinguish

FIG. 61.

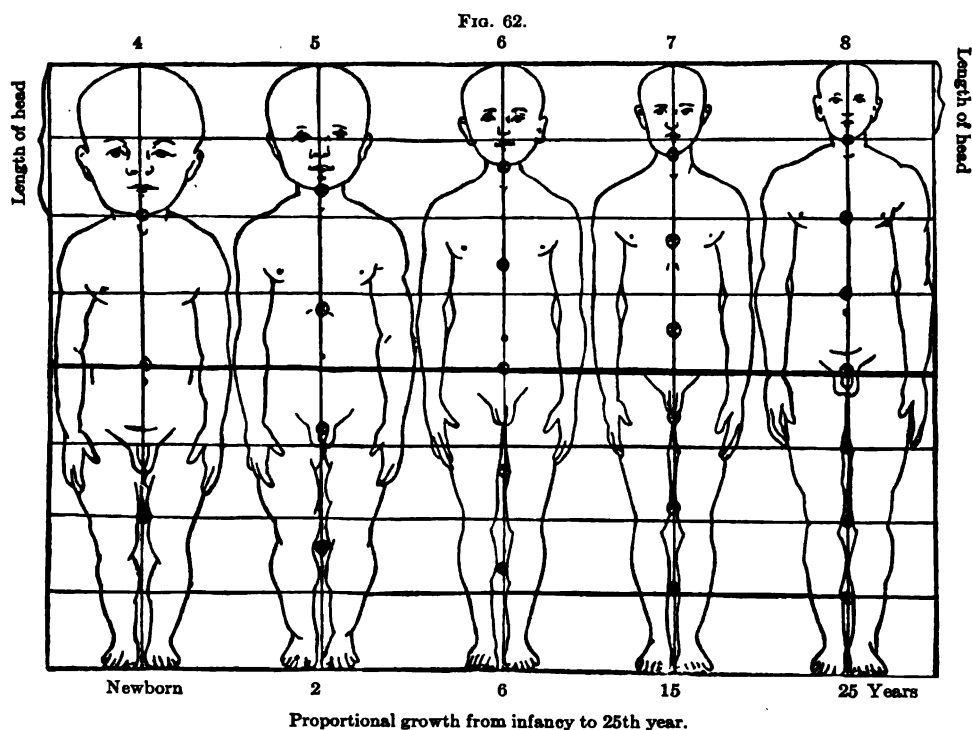


two periods of greater intensity of growth. The one occurs in the first year of life, the second in girls from the twelfth to the sixteenth year and in boys from the fifteenth to the eighteenth year (Fig. 61).

The curve of growth represents the picture of a double wave. The first period of growth may find its explanation in the continuance of the excessive foetal energy of growth; the second period coincides in boys and girls with the development of puberty, and it is probable that there exists a close relation between these two phenomena. The curves in Fig. 60 show distinctly the differences of the growth in weight of boys and girls (particularly of the girls from $13\frac{1}{2}$ to $16\frac{1}{2}$ years of age). Fig. 61 illustrates particularly the variations in the intensity of the yearly gain in weight.

The determination of the total length of the body is best adapted to investigate the growth in length, since it results from the length of the legs, the pelvis, the spinal column, and the head, and expresses most distinctly the changes in the growth of all these parts. At the same time

we obtain information about the length of the single parts of the body, since this stands in a certain relation, although this relation is not quite constant for the different years of life. For instance, in the child going to school the length of the lower leg is stated to be 28 to 29 per cent. of the height, that of the upper leg 28 to 31 per cent.; the corresponding figures for the lower arm and hand are 25 to 27 per cent., for the upper arm 18 to 19 per cent. These figures can only be regarded as approaching the average values, and only for the school period. From the following it may be seen how much these single proportions change during growth. The total length of a newborn baby is 4 times the length of the head, that of a two year old boy 5 times the height of



the head, that of a boy six years of age 6 times the length of the head, that of a fifteen year old boy 7 times the length of the head, and that of the adult eight times the height of the head. These proportions can best be seen in Fig. 62, taken from the book by Stratz, *Der Körper des Kindes*.

Numerous observations, chiefly made in maternity hospitals, inform us about the length of the body at birth. This length is 50 to 52 cm. in boys and 49 to 51 cm. in girls. At the end of the first year of life the total length reaches 70 to 75 cm. Only a few investigations have been conducted concerning the more exact course of the growth in length during the first year, probably because an exact determination of the

length is very difficult in the infant. In order to accomplish this the infant has to be placed on a table in such a way that its head touches a board fixed vertical to the plane of the table. Then one person has to hold head, neck, and shoulders in the proper position, a second person

FIG. 63.

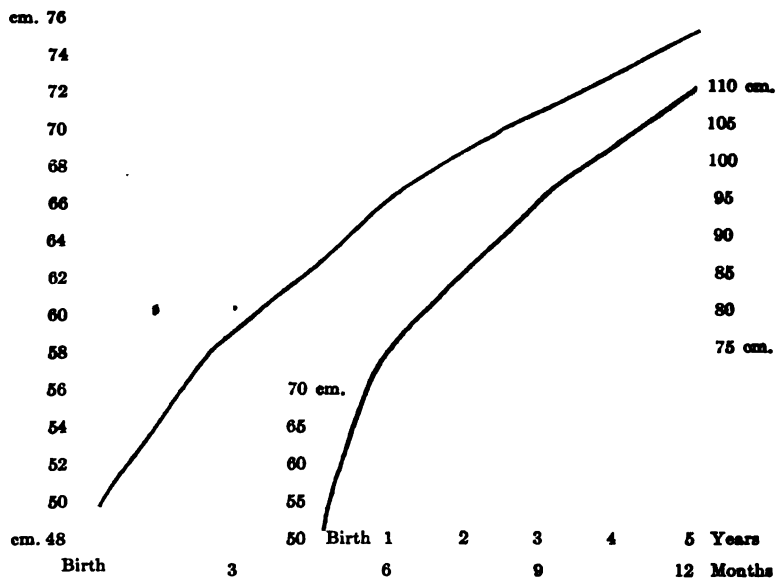


Shape of skull from left occipito-anterior position.

has to do the same with regard to pelvis and knees, while a third person places a board on the soles of the feet vertically to the plane of the table. After the baby has been placed successfully in a good position it is removed, the position of the lower board is marked, and the distance of the mark from the upper board is measured. It is true that the position in which the head, pelvis, knees, and soles of the feet are placed is arbitrary to a certain extent, but single measurements repeated at short intervals vary only a few millimetres, and the observers soon become

accustomed to placing the body in a certain position, which will be nearly the same at the individual measurements.

FIG. 64.



Growth in length during the first year (red). Growth in length during the first five years (blue).

Measurements which I conducted in this manner have shown that the length of the body does not increase during the first three weeks when compared with the length at birth. Frequently even a decrease was noted. I was able to prove that the head of infants born at full

term becomes somewhat elongated during parturition, which explains this remarkable phenomenon. Furthermore, a cephalæmatoma is frequently formed (Fig. 63, p. 420). These changes disappear gradually during the first three weeks, but they render it impossible to form an exact picture of the growth in this time. Since the deformity of the head was disregarded, the usual data about the length of the newborn exceed the true values by 1 to 2 cm., and the actual length of newborn boys must be assumed to be about 49 cm., and that of newborn girls 48 cm.

At the end of the third month of life the infants are about 9 cm. longer than at birth; in the second quarter the length increases 8 cm.; and in the third and fourth quarters 3 to 4 cm. each. Thus the total gain in length is about 24 cm. during the first year of life. With regard to the growth in length during the first year (see Fig. 64, red curve). During the second year boys and girls grow about 10 cm., during the third year 7 to 9 cm., during the fourth year about 5 cm., and at the end of the fourth year the total length reaches about 95 to 100 cm. (see Fig. 64, blue curve). From this time on the yearly increase in the length of boys is rather constantly 5 cm. until about the thirteenth year; the next three years it rises to 6 to 7 cm., and then drops rapidly. Girls gain about 4 to 5 cm. yearly from the fifth to the twelfth year; in the thirteenth and fourteenth years the yearly gain increases to 6 to 7 cm., and decreases then rapidly. The growth in length is chiefly finished after the fifteenth year in girls and after the seventeenth year in boys. The latter can be seen particularly well on the side curve of Fig. 67. This curve is from continuous observations on twenty cadets from the fourteenth to the nineteenth year, and shows that after the seventeenth year the length did not increase more than 3 cm. The course of the growth in length is laid down in the following table:

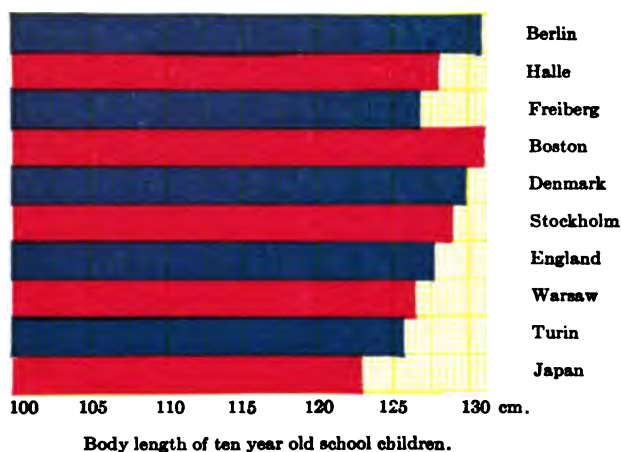
TABLE 3.

Year of life.	Boys.		Girls.	
	Length cm.	Yearly gain cm.	Length cm.	Yearly gain cm.
Birth	50	..	49	..
1	75	25	74	25
2	85	10	84	10
3	98	8	92	8
4	99	6	98	6
5	104	5	103	5
6	109	5	107	4
7	115	6	113	6
8	120	5	118	5
9	125	5	123	5
10	130	5	128	5
11	135	5	133	5
12	140	5	139	6
13	145	5	146	7
14	151	6	153	7
15	157	6	158	5
16	164	7	160	2
17	168	4	161	1
18	170	2	...	..

Growth in length.

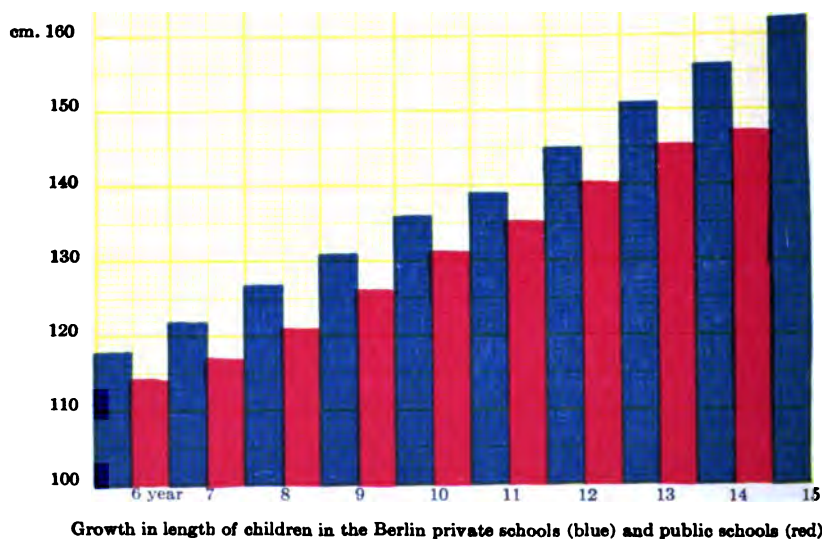
At first sight it seems surprising that the growth in height stops at the given times, since usually the limit is reached in the twenty-third to twenty-fifth year. And actually a small increase in length is nearly

FIG. 65.



always observed after the fifteenth to eighteenth year of life, and a number of boys and girls will even gain considerably in height after this time. But such growth must be regarded as delayed by previous dis-

FIG. 66.

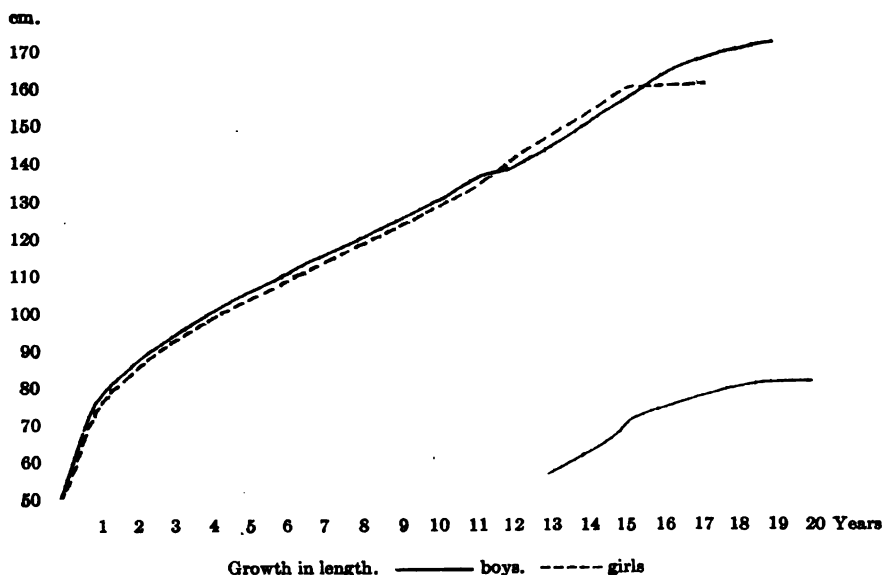


turbances. German recruits twenty years of age (Fig. 65) measure on an average 169 cm., while Germans thirty-one years of age measure 169.5 cm. Since a number of recruits (namely, those whose growth was

delayed) increase considerably in height after the twentieth year (Fig. 66), there must be many men who do not grow any more after the twentieth year, because the average difference in length between the twentieth and the thirty-first year is only 0.5 cm. In other nations, the differences for the given ages are surprisingly small; in England and France, for instance, the difference between recruits and men thirty-five years of age is 1 cm. The investigations of Ammons furnish exact figures for the population of Baden. His statistics include over 30,000 persons fit for service in the army. Among other data he found that the average height of the recruits in Baden is 165 cm.; it is therefore considerably lower than the average of a large part of the other recruits in Germany.

Racial and social conditions are of influence on the growth in height

FIG. 67.



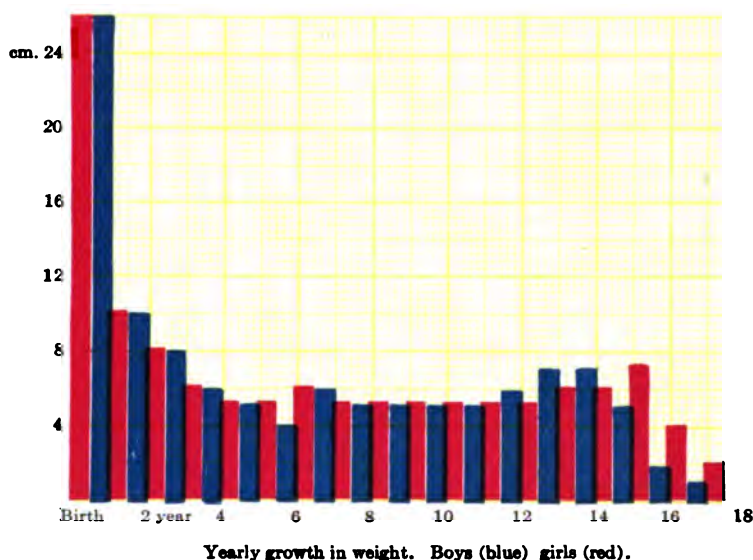
and this is shown in Figs. 65 and 66. Fig. 65 shows that considerable differences in growth exist in the different regions of Germany, and still more pronounced are these differences in comparing different countries. In Fig. 66 we see that pupils belonging to well-to-do classes present a better growth in height than do pupils coming from socially lower classes, although it is probable that this difference may change in favor of the latter after the 15th year.

The growth in height like the growth in weight, presents two periods of greater intensity, the one during the first year of life, the second after the twelfth year in girls and after the thirteenth year in boys. The reasons are undoubtedly the same as for the growth in weight. It is of interest that the curve of growth from the second to about the twelfth year is somewhat like a parabola. From the fact that during the first

half year of life the gain in length is as great as that of three to four years together later on, we may obtain some information about the strength of the impulse of growth in the first months of life. The curve of growth in girls crosses that of the boys at about the twelfth to the fourteenth year. This is occasioned by the fact that in girls the second period of growth occurs considerably earlier than in boys. After a short time the curve sinks again below that of the boys. It is therefore only from about the twelfth to the fourteenth year that the girls surpass the boys in length for a short time. Figs. 67 and 68 show these conditions graphically.

The length of human beings depends chiefly on the size of their skeletons, and in close connection to this stands the size of the muscles.

FIG. 68.



The size of the skeleton and of the muscles must exercise a considerable influence on the gain in weight of the body, since the combined weight of skeleton and muscles form 40 per cent. of the total weight of the newborn and 60 per cent. of that of the adult. The size of the skeleton and muscles has a dominating influence on the growth in length. It was to be expected, therefore, that the curves of the growth in weight and length would follow a similar course. This is actually the case, but a remarkable difference is to be noted, in that the second period of greater intensity of growth occurs somewhat earlier with regard to the length than to the weight, and that the growth in length is finished somewhat earlier than is the growth in weight.

NUTRITION AFTER THE FIRST YEAR

BY

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DR. SAMUEL AMBERG, BALTIMORE, MD.

THE determination of the quality and quantity of food required is more difficult in the case of the child than in that of the adult. The age of the child constitutes an important factor to be taken into consideration, aside from the disturbing influences which bad education and bad habits sometimes raise as obstacles to the advice of the physician. Quite generally, the principle laid down by Rubner, that the food requirement per unit of weight is proportional to the relative size of the body surface, is as valid here as in infancy. Older children take absolutely more food than younger children, but relatively the amount of food required decreases with advancing age in accordance with the decreasing proportion between the weight and the surface of the body.

Numerous and laborious investigations, following the lead of the classical work of Voit concerning the food requirement of the adult, were conducted in order to determine the food requirement of the healthy normally developed child at the different periods of its age, and, further, to discover the proper proportions of the individual food constituents. The work of Camerer particularly has been carried on with admirable consistency and exactness.

Tabulating the average values from all the investigations, considerable variations are noted, as may be seen from the following table:

TABLE 1.

Age in years.	Proteid, grams.	Fat, grams.	Carbohydrates per diem grams.
2-4	40-64	32-62	110-205
5-7	50-58	30-43	145-197
8-10	60-68	30-70	220-250
10-11	68-86	44-85	211-270

Food requirement per kilo body weight.

These apparently very great differences disappear if we calculate in each case the calorimetric values for the individual substances of the food and compare the sums of the combustion values, keeping in mind the fact that fat and carbohydrates may replace each other within wide limits.

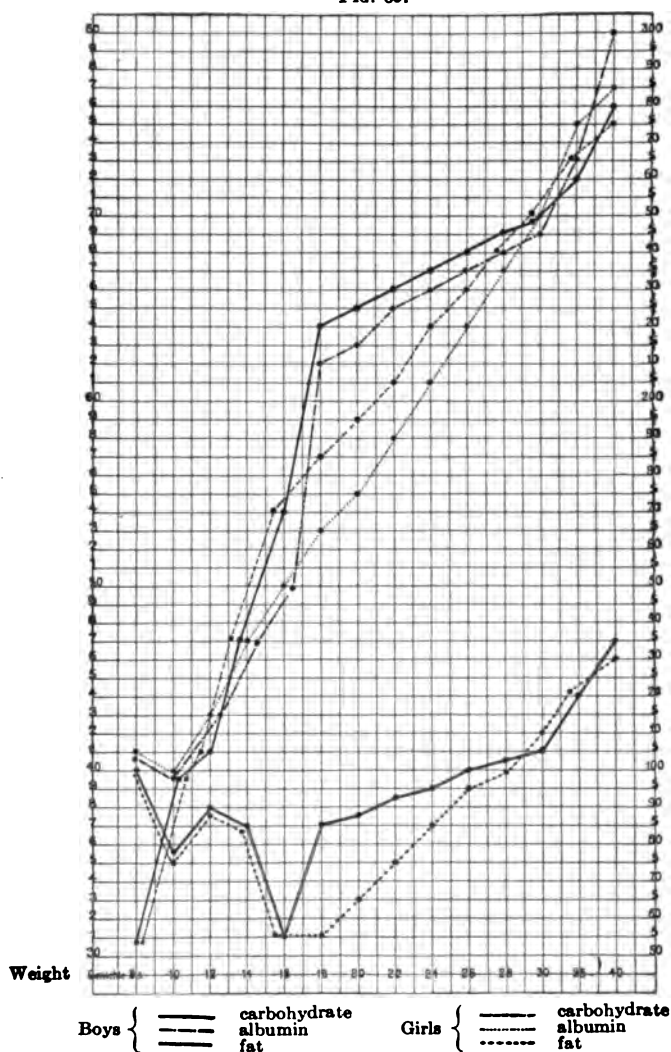
The observation of Camerer is correct, however, that children of different ages, but of about the same weight, require the same amount of food per kilo weight. The physician obtains information sufficiently

exact for practice from the table calculated according to Camerer's data with regard to the amount of proteid, fat, and carbohydrate required for 1

TABLE 2.

Age in years.	Sex.	Total amount of food.	Water.	Proteid.	Fat.	Carbohydrates.	Calories.
2-4.....	Boys.....	93.1	75.3	3.6	3.1	9.2	75.3
5-7.....	Boys.....	84.4	67.4	3.0	1.9	10.7	69
	Girls.....	84.3	66.6	3.5	2.5	10.9	76.6
7-10.....	Boys.....	75.5	59	2.7	1.3	9.9	59.2
	Girls.....	70.8	55.5	2.8	1.3	10.4	61.6
11-14.....	Boys.....	54.0	41.4	2.1	1.4	8.4	51.4
	Girls.....	56.1	44.4	2.5	1.0	7.7	47.3

FIG. 69.



The weight for all food elements is given on the horizontal line. The right vertical line gives the amount in grams for albumin and fat, the left for the carbohydrates.

kilo body weight. It is only necessary to multiply the figures then for the different periods of age by the total weight of the child in kilos.

The investigations of Camerer and others furnish the following important results: the rational proportion of animal to vegetable food is 1:2.2, that of the nitrogen containing food to that free from nitrogen 1:4 to 1:5. Generally at least 50 per cent. of the proteid introduced should be of animal origin; more specifically, the proteid was derived as follows:

TABLE 3.

From	2-4 years.	5-7 years.	8-10 years.	11-14 years.
Milk, per cent.....	42	28	18	9
Meat and egg, per cent.....	36	39	42	45
Vegetables and leguminous plants, per cent.....	22	33	40	46

Special amounts of food for the different periods of age are given in Table 4. When the food is divided into five meals, about 30 per cent. of the total amount of food is given at the noon-day meal, 25 per cent. with the supper, and the rest with breakfast and the afternoon meal (Camerer).

TABLE 4.

Kind of food.	2 years.	3 years.	4-7 years.
Milk, c.c.....	1000	1000	1000
Meat, Gm.....	60	75	100
In sandwich, Gm.....	40	50	50
Egg.....	1	1	1
Bouillion } c.c.....	100	125	200
Soup }.....	100	125	200
Vegetables, Gm.....	50	100	150
Potatoes, Gm.....	35	50	75
Rolls, Gm.....	50	125	250
Butter, Gm.....	10	15	30
	<i>Total:</i>	<i>Total:</i>	<i>Total:</i>
Proteid, Gm.....	68	79	95
Fat, Gm.....	66	75	92
Carbohydrates, Gm.....	78	123	198
Calories.....	1200	1525	2060

Amounts of food for healthy children given in five meals pro die (Steffen).

The nutrition of children that are ill or in a state of convalescence is more complicated. In addition to the factors already mentioned, the kind and severity of the disease has to be taken into consideration. Naturally, certain diseases require a special nutrition, but these special or specific forms of diet belong to the therapy of the disease and have to be considered in connection with the different diseases. In general, the nutrition of the sick child must be such as to insure a continuous gain in weight after the loss sustained during the disease has been restored. The nutrition should not only lead to a gain of fat but to a formation of muscle.

Taking all these factors into consideration, Baginsky established the following forms of diet for sick and convalescent children of different periods of age:

I. Diet for convalescents from severe diseases and for afebrile patients suffering with wasting (surgical) affections and for those suffer-

ing with chronic afebrile diseases. The first group of this subdivision includes children in whom overnutrition is the aim, the second group includes children who probably have a nearly normal physiological requirement of food.

II. Diet for patients with a moderate rise of temperature, or for those who are just recovering from a febrile disease and have entered the state of convalescence. Generally, this division includes cases which have not as yet regained their normal digestive power.

III. Diet for febrile cases, chiefly liquid food.

These three forms have to be adapted to the given period of age with regard to the amount of food, and it will be best to do this with consideration of the following limits:—(A) Children from nine to fourteen years of age; (B) Children from five to nine years of age; (C) Children from one and a half to four years of age.

In general it may be stated that children about two to four years of age require a daily amount of food made up of 50 Gm. proteid, 50 Gm. fat, and 140 Gm. carbohydrates, corresponding to about 1250 calories; children from five to eight years need about 80 Gm. proteid, 220 Gm. carbohydrates, and 65 Gm. fat, corresponding to 1825 calories; and children from eight to twelve years need 85 Gm. proteid, 275 Gm. carbohydrates, and 80 Gm. fat. These figures, given within very wide limits of age, may be tabulated for the single forms of diet as follows:

TABLE 5.

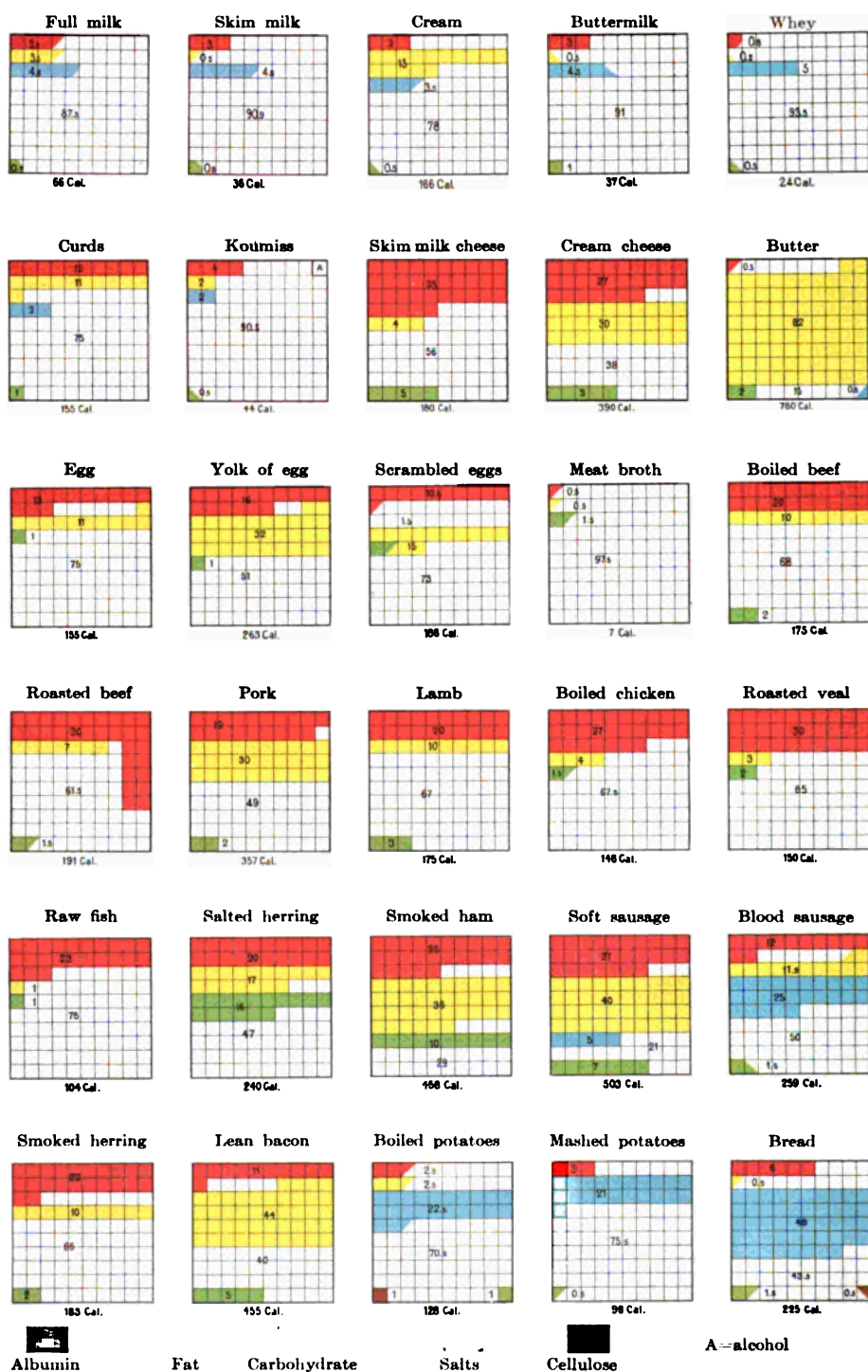
Age.	Form	Albumin.		Fat.		Carbohydrates.		Calories.		Proportion of nitrogenous to non-nitrogenous.	
		Per day.	For 1 kg. body weight.	Per day.	For 1 kg. body weight.	Per day.	For 1 kg. body weight.	Per day.	For 1 kg. body weight.	Diet. ¹	Calories.
(A) 9th-14th year.	I	85.5	3.1	82.1	3.3	271	10.8	2225	87.7	1:4.9	1:5.4
	II	80.9	3.2	85.3	3.4	250	10	2213	85.8	1:5.6	1:5.4
	III ²	69.3		72.8		95		1350		1:3.9	1:4
(B) 5th-8th year.	I	70	3.4	61	3.1	221	10.6	1570	85.8	1:5.2	1:3.5
	II	76	4	99	4.1	244	12.7	2130	106	1:6	1:4.7
	III ²	53		58		69		1040		1:4	1:4
(C) 2d-4th year.	I	51.3	4	51	3.9	149	11.4	1307	101.2	1:5.3	1:4.5
	II	43.5	4.5	44	4.5	115	11.8	1060	108.7	1:5	1:5
	III ²	42		45		49		790		1:3.8	1:4

The values fairly approach those given for healthy children with regard to the proteid, while the values for fat and carbohydrates exceed the normal ones rather considerably. The high figures for the second form find their explanation in the large amounts of milk or milk with cereals contained in this form. It is a fact frequently observed that the

¹Fat replaced by an isodynamic quantity of carbohydrates.

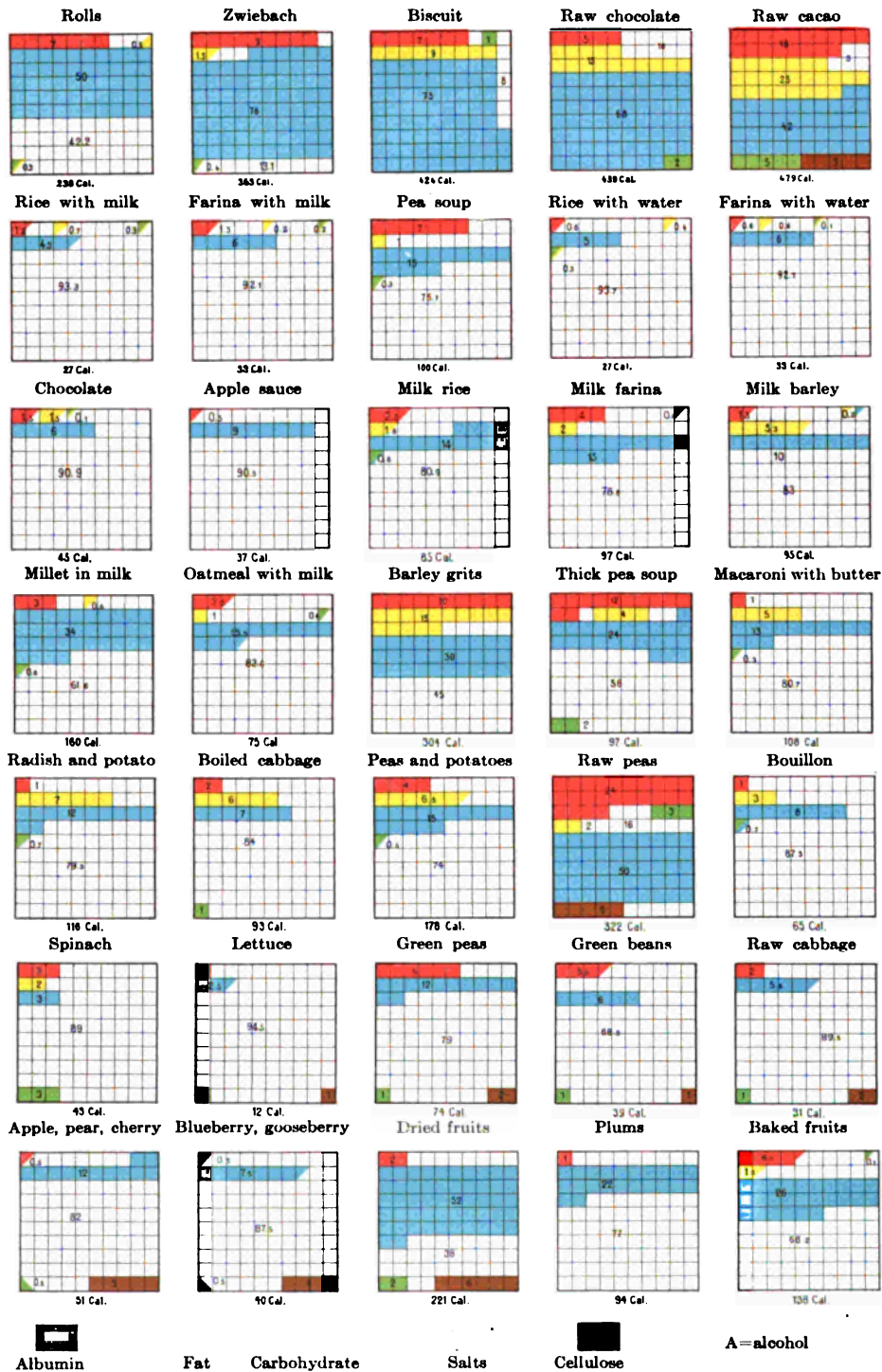
²Fever diet.

PLATE 4.



Average composition of some of the more common foods.
Graphically portrayed as percentages of the total weight.
Each large square contains 100 small divisions.
(The caloric value is estimated.)

PLATE 5.



important foods employed in childhood.
 after Gurgensen's method.
 sions=100%. Each small square=1%.
 imated on 100 gm.)

TABLE 6.

	Form I.	Form II.	Form III.
First breakfast	Milk and rolls or swieback or oatmeal gruel.	As in Form I.	Milk.
Second breakfast	Milk, rolls or soft bread with butter, and sliced meat (ham, sausage, cold roast, or egg).	Milk, egg, roll with butter.	Milk, egg.
Neon-day meal	Meat broth (clear or milk addition) or soup prepared with vegetables or with fruit, or milk or chocolate. (Barley, rice, noodles, oatmeal, grain, tapioca.) Green vegetables or cereals, or cereals with milk, dumplings with fruit, or vegetables boiled with potatoes and meat. Meat boiled or roasted, or boiled given with the vegetables. Fruit sauce, or pastry with fruit juice or fresh fruit.	Meat broth with egg, or milk soup with sliced meat (barley, rice, oatmeal). Cereals with milk, mashed potatoes. Finely chopped meat boiled or roasted. Fruit sauce, or pastry with fruit juice, fruit.	Meat broth with egg.
Afternoon meal	Milk with roll or swieback.	Milk with roll or swieback.	Milk.
Supper	Milk or milk soup, roll or soft bread with butter (sandwich) and sliced meat or egg.	As in Form I.	Milk or milk soup.

TABLE 7.
PERIOD OF AGE.

			(A)			(B)			(C)		
			I	II	III	I	II	III	I	II	III
First breakfast.	Milk or oatmeal gruel	e.c.	500	333	500	500	333	333	250	250	250
	Roll	Gm.	100	100	...	100	50	...	50	...	...
	or Zwieback	Gm.	...	...	...	80	40	...	40	40	...
Second breakfast.	Bread	Gm.	120	...	...	50	...	...	50	...	...
	or Roll	Gm.	100	50	...	...	50	...	50	50	...
	Sliced meat (ham, sausage)	Gm.	30	...	...	30	...	...	10	...	...
	Butter	Gm.	30	15	...	15	10	...	10	10	...
	Eggs	...	...	1	...	...	1	...	...	1	...
Neon-day meal.	Milk	e.c.	500	333	500	333	333	333	250	250	250
	Meat broth or soup	e.c.	250	250	333	250	250	250	125	125	125
	Hacked roast	Gm.	...	75	...	...	75	...	50	50	...
	Meat (boiled or roasted)	Gm.	150	...	...	125	...	...	...	...	...
	Eggs	Gm.	...	...	1	...	...	1	...	...	1
	Vegetables (green)	Gm.	666	...	...	333	...	...	...	...	...
	Cereals with milk	e.c.	...	333	...	...	250	...	...	125	...
Afternoon meal.	Potato (as addition)	Gm.	250	...	...	...	...	...	...	...	...
	Fruit sauce	Gm.	25	...	...	...	...	...	...	...	...
	Milk	e.c.	500	333	500	333	333	333	250	250	250
	Roll	Gm.	100	100	...	50	50	...	50	50	...
Supper.	or Zwieback	Gm.	...	...	...	...	40	...	40	40	...
	Milk	e.c.	400	333	...	250	250	...	250	250	250
	or Milk soup	e.c.	...	...	333	...	...	333	...	...	...
	Eggs	Gm.	1	1	...	1	1	...	1	...	...
	Bread	Gm.	120	...	...	...	...	...	...	...	...
Total of calories	or Rolls	Gm.	...	50	...	50	50	...	50	50	...
	Sliced meat	Gm.	30	...	...	30	...	...	...	...	...
Total of calories			3200	2900	1800	2000	1800	1100	1400	1000	800

children from the poorer classes possess a great liking for carbohydrates; this liking must be taken in consideration, as our material is obtained chiefly from the poorer classes. Table 6 gives a review of the qualitative composition of the single forms of diet, and Table 7 gives the proportional amounts of these forms for the different periods of age and for the single meals.

These tables give the widest scope for the choice of individual food materials. The success of the feeding will always be dependent to a large extent on the skilful choice and assortment of the dishes. The foundation and first condition of a diet is a definite minimal amount of proteid, fat, and carbohydrates capable of sustaining the body and insuring a gain in weight. The manner in which this is to be accomplished offers a large and grateful field for the skill of the thinking physician. The psychic influences of the nutrition must not be disregarded, as these are of great importance in making the child thrive.

In this connection, the value of proprietary foods for the nutrition of children has to be discussed. Within the last decade a large number of such preparations have appeared, and in the most varied forms. They are not to be admitted to the bill of fare of the healthy child, and have no field in purely physiological nutrition. They are superfluous, to say the least, and sometimes they are actually injurious if wrongly used by ill-advised parents. In addition, the most of them are very expensive.

The pediatricist would not like to dispense with these preparations under certain pathological conditions, during convalescence, and in certain special cases. Their use will have to be considered in the special chapters. But we may call attention to the fact that the most expensive preparations with high sounding names are not always to be preferred to the cheaper ones.

The ready-made dishes tabulated in Plates 4 and 5 were prepared as follows:

1. Scrambled egg: 3 eggs, butter 15 Gm., milk 65 c.c., to make total of 200 c.c.
2. Rice soup with milk: Rice 70 Gm., milk 540 c.c., water 540 c.c.
3. Barley soup with milk: Barley 70 Gm., milk 540 c.c., water 540 c.c.
4. Rice soup with water: Rice 140 Gm., butter 12 Gm.
5. Barley soup with water: Barley 140 Gm., butter 12 Gm.
6. Chocolate soup: Chocolate 18 Gm., flour 26 Gm., milk 540 c.c., water 540 c.c.
7. Milk rice: Rice 200 Gm., milk 400 c.c., water 400 c.c., butter 20 Gm., sugar 25 Gm.
8. Milk barley: Barley 75 Gm., milk 350 c.c., water 400 c.c., butter 30 Gm., sugar 30 Gm.
9. Milk grain: Grain 120 Gm., milk 450 c.c., water 700 c.c., butter 45 Gm., sugar 30 Gm.
10. Milk millet: Millet 100 Gm., milk 500 c.c., water 500 c.c., butter 20 Gm., sugar 20 Gm.
11. Grits with milk: Grits 130 Gm., milk 600 c.c., water 700 c.c., butter 20 Gm., sugar 20 Gm.

12. Purée of leguminous vegetables: Vegetable 500 Gm., butter 40 Gm.
 13. Spinach: Spinach 750 Gm., bouillon or water 400 Gm., butter 20 Gm., (and sugar 20 Gm.).
 14. Carrots with potatoes: Carrots 480 Gm., potatoes 100 Gm., meat broth 400 c.c., butter 20 Gm.
 15. Leguminous vegetables with potatoes: Peas 125 Gm., potatoes 100 Gm., meat broth 400 c.c., butter 20 Gm.
 16. Oatmeal with bouillon: Oatmeal 140 Gm., bouillon 550 c.c., water 550 c.c.
 17. Baked fruit with dumplings: Baked fruit 200 Gm., 1 egg, flour 280 Gm.
- Nos. 2 to 17 are for a finished dish of about 1000 Gm. each.

COMPOSITION OF URINE AND FECES AFTER THE FIRST YEAR OF LIFE

TRANSLATED BY
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URINE

THE quantity of urine voided by children after infancy is, because of numerous observations, known with some exactness. The quantity does not increase with age and weight like the food, as Camerer supposes, but more slowly; particularly in boys. But the concentration and amount of urea increase very markedly. This occurs in consequence of the very strong perspiration, which is observed in older boys who exercise much; and in them it exceeds considerably that of younger children and of adults. The following tables (Camerer) contain the average values for the different periods of age; naturally, they vary within fairly wide limits, as may be seen from a number of my own observations recorded below:

	Girls.				Boys.		
Age, in years.....	2-4	5-7	8-10	11-14	5-6	7-10	11-14
Quantity, in c.c.....	670	800	890	930	730	940	1040
Specific gravity.....	1.017	1.017	1.016	1.018	1.019	1.020	1.019
Total nitrogen in grams.....	6.3	7.2	8.3	8.9	7.6	9.3	11.7
Urea in grams.....	12.0	13.8	14.7	17.0	14.6	15.7	22.4
Urine for each 100 c.c.....							
Water of food.....	71	72	76	70	61	70	68
Urine for each 100 c.c. liquid taken....			94	91			98

Average 24 hour quantity of urine according to Camerer Sr.

The average number of micturitions per diem is, according to Camerer:

	Age in years.	Micturitions.
Girls.....	2-4	6.5
	5-7	5.2
	8-10	4.4
	11-14	3.7
Boys.....	5-6	5.0
	7-10	5.1
	11-14	3.0

Average number of micturitions.

The cases used for the table are either convalescents from acute febrile infectious diseases, or those present in the hospital on account of surgical diseases: All cases were afebrile and expected soon to be discharged. No urine was pathological. The diet corresponds with the forms previously given.

Age and sex.	Duration of observation in days.	Diet.	Quantity of urine, in c.c.	Specific gravity.	Total nitrogen in urine.	Urine per 100 H ₂ O of the food.
2½ years, male.	8	II, C. ¹	480		6.8	60
3¼ years, male.	10	II, C.	250	1.028	4.3	30
5 years, male.	10	II, B.	480	1.019,6	4.4	60
5½ years, female.	10	II, B.	600	1.020		60
5½ years, male.	4	Much milk. Only milk (3.5 litres).	1680	1.006	14.6	64
6 years, female.	4	Only milk (2.5 litres).	1480	1.015	12.5	66
6 years, male.	10	II, B.	1190	1.015	10.36	55
7 years, male.	4	Only milk (3-4 litres.)	2260	1.011	15.4	70
7 years, male.	30	I, B.	460	1.022		40
7 years, male.	12	I, B.	656	1.018	10.13	50
7 years, female.	10	I, B.	560	1.018	5.55	60
7 years, male.	10	I, B.	680	1.021	7.13	56
8 years, male.	4	II, B.	1500	1.014	18.4	60
9 years, male.	10	II, A.	1110	1.017	10.93	60
10 years, female.	10	II, A.	420	1.020	5.15	60
11 years, male.	10	I, A.	1100	1.015,6	10.49	64
11 years, female.	12	I, A.	783	1.019	11.1	50

¹ Compare Tables 5 and 6 of the chapter on Nutrition.

The following contains some data concerning the composition of the urine of older, healthy children, with regard to the division of the nitrogen in the individual constituents.

I. Average 24 hours, excretion of two 3 year old boys, according to Camerer, Jr.

Diet: Table diet, milk, meat, vegetables, potatoes, egg, bread, much water.

II. Average values of 4 girls 3 years of age. *Diet:* 1 litre milk and little table diet (Camerer, Jr.).

III. Average values of the 24 hours excretion of a girl 7 years of age. Duration of observation, 10 days. *Diet:* Milk (1000), zwieback (150), egg (140), meat (100), cereals with milk (100).

IV. Average values of a boy 7 years of age. Duration of observation, 8 days. *Diet:* Milk (1000), rolls (150), butter (25), vegetables (100), bouillon (250), meat (125).

V. Average values of a 7 year old girl. Duration of observation, 8 to 9 days. *Diet:* Like III.

VI. Average values of an 11 year old girl. Duration of observation, 12 days. *Diet:* Rolls (250), butter (35), vegetables (200), meat (125), milk (1000), cocoa (30), bouillon (250).

A few remarks may be added. The secretion of uric acid varies very much in the older child, as the investigations of Göppert show. A normal value cannot be stated. If the kind and amount of food are equal, the proportion of uric acid to the total nitrogen of the urine remains constant. This proportion depends on the quality of the food, but not on the amount of nitrogen introduced. The preparation of sul-

furic ether acids to the total sulfuric acid is very constant, but its absolute amount is subject to wide variations. The mineral constituents of the child's urine do not differ materially from those of the adult; unlike the urine of the infant, the sodium chloride forms the main part of the salts in the older child.

Number.	Quantity of urine.	Specific gravity.	Total nitrogen.	Urea group.			Purin group.			Total P_2O_5	P_2O_5 of the acid salts.	Observer.
				Urea nitrogen.	Ammonia nitrogen.	Total nitrogen.	Uric acid nitrogen.	Base nitrogen.	Total nitrogen.			
I	505	1.016,5	4.115	3.495	0.315	3.810				0.695	0.38	Camerer, Jr. Camerer, Jr. Sommerfeld.
II	470	1.022,5	5.20	4.80	0.29	5.09	0.065	0.013	0.078	1.08	0.64	
III	620	1.019	5.15	4.562	0.330	4.892	0.070		0.070	1.36		
IV	808	1.015	10.13	8.864	0.560	9.424	0.078		0.078			Sommerfeld.
V	133	1.016,8	6.16	5.360	0.449	5.809	0.0703		0.0703	1.25		Sommerfeld.
VI	940	1.019	11.1	9.702	0.750	10.452	0.0796		0.0796			Sommerfeld.

FECES

The feces of children beyond the age of infancy are not different from those of the adult. Mixed diet is well utilized and with increasing age the utilization becomes still better, since the absolute (in girls the

	Age, in years.	Quantity, in grams.	Solids, in grams.	Nitrogen, in grams.	Acid ether extract, in grams.
Girls	2-4	72	16	1.1	3.6
	5-7	67	15	1.0	2.9
	8-10	70	15	1.2	3.1
	11-14	84	18	1.3	3.3
Boys	5-6	134	28	2.1	3.4
	7-10	113	23	1.8	3.4
	11-14	98	23	1.3	5.2

Twenty-four hours' averages for the two sexes, according to Camerer.

	Age, in years.	Total acids.	Acid ether extract.	Ash.	Nitrogen.
Girls	2-4	5	16	21	12
	5-7	6	8	25	14
	8-10	5	10	19	12
	11-14	4	10	17	11
Boys	5-6	8			18
	7-10	6	9	22	15
	11-14	5	14	18	10

Resorption (showing composition of feces, in grams per 100 Gm. of food, according to Camerer).

relative) daily amount of feces and the number of passages decreases (Camerer). The following tables give an idea of the composition of the feces and of the utilization of fat and nitrogen.

The high ash content during the early age periods is the result of an abundant milk diet. Feces after feeding on cow's milk contain about 8.7 per cent. ash. Finally, some further data may be given of the re-

	Age, in years.	Solids.	Nitrogen.	Acid ether extract.	Ash.
Girls	2-4	22	1.5	4.9	4.4
	5-7	22	1.4	4.3	4.1
	8-10	22	1.6	4.5	3.3
	11-14	22	1.5	4.5	2.7
Boys	5-6	21	1.6		
	7-10	20	1.6	2.9	3.1
	11-14	24	1.3	5.8	2.8

Content in grams of 100 gm. of fresh feces, according to Camerer.

sults of my own and other investigations with regard to the utilization of the fat, nitrogen, and solids of the feces. The observations of Rubner, Camerer, Söldner, Preussnitz, and others, more recently confirmed by

Age (in years) and sex (m-male, f-female).	Kind of food.	Solids of food, per gram.	Solids of feces, per gram.	For 100 solids of the food, solids in feces.	Nitrogen of food, in grams.	Nitrogen of feces, in grams.	Minimum amount assimilated.	Fat of food, in grams.	Fat of feces, in grams.	Therefore assimilated.	Investigator.
1½	Milk	...	...	...	...	...	93 %	...	...	94.9%	Bendix.
2	Bread....	...	...	...	...	...	91 %	...	...	91 %	Bendix.
2½	Milk	...	...	...	...	...	84 %	...	...	91 %	Bendix.
2½	Bread....	...	...	...	...	...	84 %	...	...	91 %	Bendix.
2½	same first chocolate	...	...	...	...	...	84 %	...	...	91 %	Bendix.
2½ m.	I, C.	201	13.25	6.6	9.602	0.604	8.998 =94.7%	...	...	...	Sommerfeld.
3½ m.	I, C.	208	8.61	4.2	6.96	0.54	6.42 =91.8%	44.2	6.3	37.9 =85.6%	Sommerfeld.
5 m.	II, B.	251	11.94	4.8	8.224	0.815	7.429 =90.2%	51	3.28	47.64 =93.6%	Sommerfeld.
5½ m.	Milk only.	360	24.41	6.8	16.39	0.965	15.425	...	...	...	Sommerfeld and Caro.
6 f.	Milk only.	308	31.21	10	13.69	0.585	13.155	...	...	...	Sommerfeld and Caro.
6 f.	I, B.	307	17.13	5.6	12.21	1.19	11.02 =90.2%	99.25	3.19	96.06 =96.8%	Baginsky and Sommerfeld
7 m.	Milk only.	484	33.42	6.9	19.98	1.06	18.92	...	...	...	Sommerfeld and Caro.
7 m.	I, B.	302	13.93	4.6	11.23	0.997	10.233 =91.1%	60.98	3.29	57.69 =94.7%	Sommerfeld.
7 m.	I, B.	308	24.6	8	12.895	2.025	10.870 =84.3%	59.92	3.46	56.49 =94.3%	Sommerfeld.
8 m.	II, B.	200	20.72	10.3	14.942	1.024	13.918 =93.1%	...	...	...	Sommerfeld.
9 m.	II, A.	416	30.02	7.2	12.976	1.49	11.488 =89 %	85.37	3.29	82.08 =96.1%	Baginsky and Sommerfeld
11 m.	I, A.	835	19.3	5.8	14.18	0.87	13.31 =93.8%	82.10	3.43	78.67 =95.8%	Sommerfeld.
11 f.	I, A.	308	44.9	14.47	14.11	2.70	11.41 =81.7%	73.8	4.15	69.65 =94.4%	Sommerfeld.

Caro and myself, show that exclusive milk diet is utilized very well by older children (up to 4 litres a day), very much better than by the adult (see data on the table).

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